CLINICAL SCIENCE

THE IMPACT OF OBESITY ON PULMONARY FUNCTION IN ADULT WOMEN

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INTRODUCTION: Obesity can cause deleterious effects on respiratory function and impair health and quality of life.
OBJECTIVE: To evaluate the effects of obesity on the pulmonary function of adult women.
METHODS: An obese group, constituted of 20 women between 20 and 35 years old with a BMI of 35 - 49.99 kg/m^2 who were non-smokers and sedentary and had no lung disease were recruited. The non-obese group consisted of 20 women between 20 and 35 years old who were sedentary and non-smokers and had no lung disease and a body mass index between 18.5 and 24.99 kg/m^2. Spirometry was performed in all subjects. The statistical analysis consisted of parametric or non-parametric tests, depending on the distribution of each variable, considering $p < 0.05$ to be statistically significant.
RESULTS: The obese group presented a mean age of 25.85 ± 3.89 years and a mean BMI of 41.1 ± 3.46 kg/m^2, and the non-obese group presented a mean age of 23.9 ± 2.97 years and a mean body mass index of 21.91 ± 1.81 kg/m^2. There were no significant differences between the obese group and the non-obese group as to the age, vital capacity, tidal volume, forced vital capacity, and forced expiratory volume in one second. However, the obese group presented a greater inspiratory reserve volume (2.44 ± 0.47 L *vs.* 1.87 ± 0.42 L), a lower expiratory reserve volume (0.52 ± 0.32 L *vs.* 1.15 ± 0.32 L), and a maximal voluntary ventilation (108.5 ± 13.3 L/min *vs.* 122.6 ± 19.8 L/min) than the non-obese group, respectively.
CONCLUSION: The alterations evidenced in the components of the vital capacity (inspiratory reserve volume and expiratory reserve volume) suggest damage to the chest mechanics caused by obesity. These factors probably contributed to a reduction of the maximal voluntary ventilation.

KEYWORDS: Body Mass Index; Chest Mechanics; Spirometry; Pulmonary Volumes; Maximal Voluntary Ventilation.

INTRODUCTION

Obesity is a chronic disease characterized by excessive body fat that causes damage to the individual's health[1,2] and is associated with comorbidities such as diabetes[3] and hypertension.[3,4] and vascular dysfunction[5,6] Obesity in adults is defined by the World Health Organization (WHO) as having a body mass index (BMI) that is greater than or equal to 30 kg/m^2.[2] The normal BMI range is between 18.5 and 24.99 kg/m.[24]

Currently, there are estimated to be one billion overweight adults, and at least 300 million of them suffer from clinical obesity.[7,8] It was recently published that, in Brazil, 41.1% of men and 40.0% of women are overweight; 8.9% and 13.1%, respectively, are obese.[9]

Obesity can cause various deleterious effects to respiratory function, such as alterations in respiratory mechanics, decrease in respiratory muscle strength and endurance, decrease in pulmonary gas exchange, lower control of breathing, and limitations in pulmonary function tests and exercise capacity.[10-14] These changes in lung function are caused by extra adipose tissue in the chest wall and abdominal cavity, compressing the thoracic cage, diaphragm, and lungs. The consequences are a decrease in diaphragm displacement, a

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decrease in lung and chest wall compliance, and an increase in elastic recoil, resulting in a decrease in lung volumes and an overload of inspiratory muscles.[11] These changes are worsened by an increase in the BMI.[15]

Since morbid obesity is always associated with various other alterations, especially those of pulmonary origin, it becomes necessary to assess the respiratory function of obese individuals. This helps to identify and treat these changes at an early stage in order to prevent negative effects on health and quality of life. Therefore, the objective of this study was, by comparing obese and non-obese subjects, to evaluate the impact of obesity on the pulmonary function of adult women with no history of pulmonary disease.

METHODS

From March to October 2007, 20 sedentary women ages 20 to 35 years old who were non-smokers, showed no history of lung diseases, had a BMI between 35 and 49.99 kg/m^2, and were on a waiting list for bariatric surgery at the Merdional Hospital, were selected to comprise the obese group (OG). To compose the non-obese group (NOG), 20 sedentary and healthy female university students aged between 20 and 35 years old who were non-smokers, presented no lung disease, and had a BMI ranging from 18.5 to 24.99 kg/m^2 were selected. Patients showing obstructive or restrictive alterations in the pulmonary function tests, obstructive sleep apnea syndrome, and an inability to perform the tests adequately were excluded. The patients signed an informed consent term, and the study was approved by the UNIMEP Ethics Committee, protocol number 68/06.

The evaluation of pulmonary function was performed by conventional spirometry using an EasyOne™ (Model 2001) computerized spirometer (ndd Medizintechnik AG, Zurich, Switzerland). The directly evaluated parameters were lung volumes, capacities, and flows through the procedures of Slow Vital Capacity (SVC), Forced Vital Capacity (FVC), and Maximal Voluntary Ventilation (MVV), performed in this order at least three times each, according to the standards of the American Thoracic Society (ATS) and the European Respiratory Society (ERS) (2005),[16] with volunteers in the sitting position. Results were expressed as absolute values and as percentages of the reference predicted values from Pereira et al.[17] (1992). By means of the SVC procedure, it was possible to obtain the following variables: vital capacity (VC), tidal volume (VT), inspiratory reserve volume (IRV), and expiratory reserve volume (ERV). The FVC procedure allowed for the determination of the forced expiratory volume in one second (FEV_1) and the FEV_1/FVC ratio. The MVV was expressed in L/min and as percentages of the reference predicted value.

Patients were questioned regarding the presence of comorbidities such as hypertension, diabetes, and dyslipidemia. Moreover, the sensation of dyspnea was evaluated in obese patients using the Medical Research Council (MRC)[18] scale, which consists of five questions based on the degrees of various physical activities that lead to breathlessness. A patient's dyspnea is classified according to the following grades: absent (0), light (1), moderate (2), moderate / severe (3), and severe (4).

The data were submitted to the Shapiro-Wilk test, and once a normal distribution was confirmed, the independent *t*-test was applied. A significance level of $p \leq 0.05$ was considered for all tests. The Pearson's correlation was used to show a relationship between the variables BMI, IRV, ERV, and MVV. The study of the sample size showed a power $\geq 80\%$ for $p \leq 0.05$.

RESULTS

As shown in Table 1, the obese and non-obese groups did not differ in age. However, the OG showed a higher BMI, as expected. In the NOG, there were no patients presenting comorbidities, while in the OG, seven patients showed dyslipidemia and four presented hypertension. None of the patients was diabetic. The dyspnea score (MRC) was 0.85 ± 0.49 (grade 0 - n = 4, grade 1 - n = 15, grade 2 - n = 1).

Concerning the spirometric variables (Table 1), there were no statistical differences between groups for the VT, VC, FVC, and FEV_1. The OG showed a higher IRV and lower ERV compared with the NOG. The MVV was lower in the OG. The groups were statistically different for the FEV_1/FVC ratio, but all were within the normal values; therefore, none of the patients showed obstructive or restrictive pulmonary disorder (Table 1).

When correlating the IRV with the BMI, a moderate positive correlation (r = 0.50) (p = 0.0011) was observed (Figure 1).

When correlating the BMI with the ERV and the MVV, a strong negative correlation with the former (r = - 0.69) and a moderate correlation with the latter (r = - 0.38) were observed, both showing statistical significance ($p \leq 0.01$) (Figures 2 and 3).

A strong positive correlation between the ERV and the MVV (r = 0.59), showing a statistical significance ($p <$ 0.0001), was also observed when both groups were evaluated (Figure 4).

DISCUSSION

Among the harmful effects of obesity to health, the respiratory changes represent an additional factor of

Table 1 - Age, body mass index (BMI), and spirometric variables of obese and non-obese groups

Variables	OBESE GROUP (n=20)	NON-OBESE GROUP (n=20)	*p*
AGE (years)	25.85 ± 3.89	23.90 ± 2.97	*ns*
BMI (kg/m^2)	41.11 ± 3.47	21.92 ± 1.82	*<0.0001*
FVC (L)	3.80 ± 0.55	3.77 ± 0.55	*ns*
%FVC	100.80 ± 11.81	98.25 ± 9.21	*ns*
FEV_1 (L)	3.10 ± 0.43	3.33 ± 0.44	*ns*
$\%FEV_1$	95.75 ± 11.16	100.05 ± 8.44	*ns*
FEV_1/FVC	0.83 ± 0.04	0.89 ± 0.04	*< 0.01*
VC (L)	3.74 ± 0.59	3.71 ± 0.64	*ns*
%VC	99.12 ± 12.96	96.45 ± 10.60	*ns*
TV(L)	0.77 ± 0.20	0.69 ± 0.25	*ns*
IRV (L)	2.44 ± 0.47	1.87 ± 0.42	*< 0.001*
ERV (L)	0.52 ± 0.32	1.15 ± 0.32	*< 0.001*
MVV (L/min)	108.50 ± 13.30	122.60 ± 19.80	*< 0.01*
%MVV	92.70 ± 9.08	102.95 ± 13.32	*< 0.01*

FVC: forced vital capacity; % FVC: percentage of predicted FVC values; FEV_1: forced expiratory volume in one second, % FEV_1: percentage of predicted VEF_1 values; MVV: Maximum voluntary ventilation; % MVV: percentage of predicted MVV values; VC: vital capacity; % VC: percentage of predicted VC values; TV: tidal volume; IRV: inspiratory reserve volume; ERV: expiratory reserve volume; *ns*: not significant.

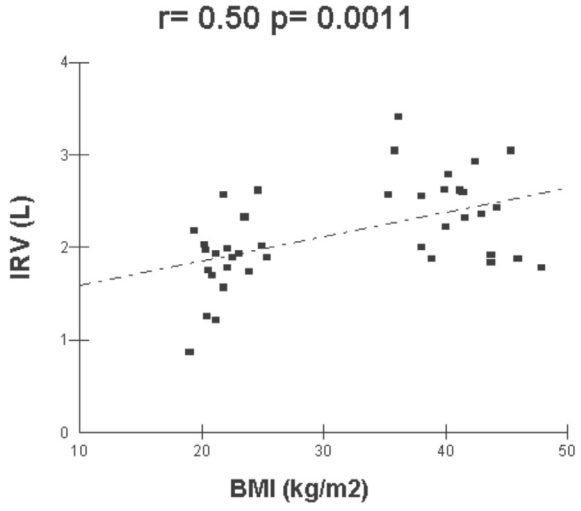

Figure 1 - Correlation between the body mass index (BMI) and the inspiratory reserve volume (IRV)

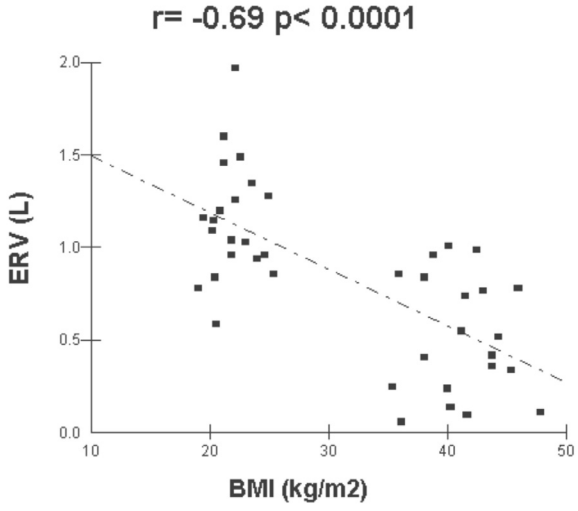

Figure 2 - Correlation between the body mass index (BMI) and the expiratory reserve volume (ERV)

functional limitation and detriment to the quality of life of obese individuals. In assessing the impact of obesity on the pulmonary function of adult women who were non-smokers and presented no history of lung disease or prior changes in the pulmonary function tests, important and significant differences were found between obese and non-obese subjects for the ERV, IRV, and MVV. According to the results, such differences can be attributed to obesity.

One of the most significant findings regarding changes in ventilation caused by obesity was the reduction in the ERV[11-15,19] confirmed by our results. A negative correlation was observed between the BMI and the ERV. Jones and Nzekwu[15] (2006) reported a reduction in the functional residual capacity (FRC), suggesting that the ERV reduces 5% per unit of increase in BMI and that, above 30 kg/m^2, it reduces 1% per unit of BMI. According to Koenig[11] (2001), this fact is attributed to the reduction of the diaphragm mobility in the chest, as the diaphragm is pressed upwards due to the expanded abdominal volume of obese individuals, a mechanical disadvantage for this muscle. Another factor

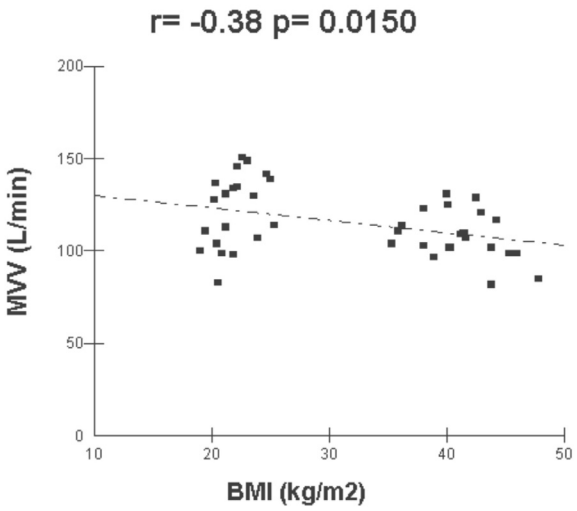

Figure 3 - Correlation between the body mass index (BMI) and the maximum voluntary ventilation (MVV)

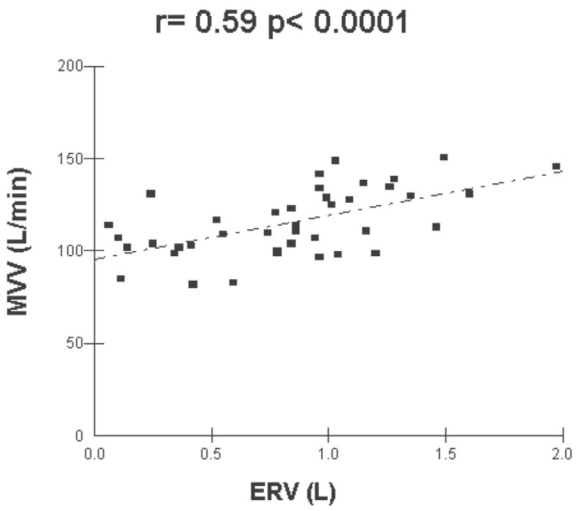

Figure 4 - Correlation between the expiratory reserve volume (ERV) and the maximum voluntary ventilation (MVV)

that may lead to the reduction in the ERV is the pulmonary compliance due to obesity.[20] Besides these detrimental mechanical aspects to the pulmonary function of obese individuals, Young et al.[21] (2003) suggested that the reduction of the ERV can lead to the increase of areas of atelectasis, harming the ventilation/perfusion mismatch and leading to arterial hypoxemia in those individuals.

Some authors have suggested that obesity may promote air trapping, which impairs adequate pulmonary ventilation through the reduction of pulmonary volumes.[12,22] Teixeira et al.[22] (2007) showed an increase in the residual volume (RV) associated with the reduction in the ERV in obese subjects who had dyspnea complaints. The authors suggested that the reduction in the ERV can be attributed to the obstruction of small airways and a consequent reduction in gas exchange. Ladosky, Botelho, and Albuquerque[12] (2001), comparing a group of obese and non-obese patients, also suggested that the reduction of the ERV may be a consequence of air trapping caused by obesity and leading to a reduction in the MVV. Although our methodology did not assess the extent of air trapping, it was observed that, in addition to the reduction in the ERV, there was also a reduction in the MVV. These variables showed a highly significant and positive correlation.

The MVV test evaluates the respiratory endurance and is influenced by the respiratory muscle strength, the lung and chest compliance, and the control of breathing and airway resistance.[12,23] In the case of obese individuals, this variable is reduced mainly by mechanical injury to the respiratory muscles, caused in particular by the excessive weight on the thorax.[12]

The increase in the IRV in obese individuals is not a common finding, but it was already reported by some authors.[11,12] Rasslan et al.[12] (2004) showed that the inspiratory capacity (IC) was higher in obese individuals than in non-obese ones, even when all the other spirometric values were within the normal range. These authors suggested that this fact may indicate normal lung compliance and an ability of the respiratory muscles to compensate, though temporarily, for the excess weight on the chest and abdomen. According to our results, no differences were observed between obese and non-obese individuals for the VC and VT. However, obese individuals showed a reduction in the ERV, possibly offset by the increase in the IRV, thus keeping the VC unchanged. These findings suggest that obesity causes injury to ventilatory mechanics (reducing the ERV) and that these changes seem to generate overload to the accessory inspiratory muscles that work to compensate the ventilation (i.e., increase IRV).

It has been suggested that obesity causes overload to the accessory respiratory muscles and that these muscles are primarily responsible for the dyspnea reported by the obese group.[13,24] El-Gamal et al.[24] (2005) found, in obese patients waiting for bariatric surgery, an increase in the respiratory drive associated with the more serious complaints of dyspnea and a reduction in static lung volumes. When assessing the same patients one year after the surgery, it was observed that the reduction in weight also reduced the respiratory drive and dyspnea complaints. This increased overload on accessory respiratory muscles caused by obesity may indirectly contribute to the increase in the IRV, thus maintaining the VC. However, the overload was not enough to cause major dyspnea in the patients studied. Other authors found a connection between obesity and dyspnea when studying a group of patients older than ours and with higher BMI levels.[22,24]

According to recent studies, the reduction in lung volume in obese people is not only directly related to the increase in the BMI; it is also related to the distribution of body fat.[10-12] Ochs-Balcom et al. (2006)[25] stated that abdominal adiposity contributes to impairment of lung function and is even more important than general adiposity markers such as weight and BMI. Once body fat is more peripherally distributed in women and more centrally distributed in men, pulmonary function is also affected according to gender, as verified by some authors.[12,17,25] Hence, only women patients were selected. New studies are needed in order to find out whether the results observed in this study can be applied to men.

CONCLUSIONS

Based on our results, we can conclude that obesity causes significant changes in respiratory function, as is evidenced by the highlighted changes in the components of the VC (IRV and ERV). These findings suggest deleterious effects on ventilatory mechanics caused by obesity, due to probable lung compression (reduction in the ERV), leading to a compensatory increase in the IRV in an attempt to maintain a constant VC. Harming the ventilatory mechanics associated with ERV reduction may have contributed to the reduction in the MVV. However, these changes were not sufficient to cause obstructive or restrictive pulmonary disorders by spirometry or significant breathlessness complaints in obese women with a BMI between 35 and 49.99 kg/m^2.

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