Original Article

Evaluation of Pulmonary Function in Class I and II Obesity*

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Background: Obesity can affect the thorax, diaphragm and abdominal muscles, thereby resulting in altered respiratory function.

Objective: To evaluate the effects of obesity and to determine whether body mass index (BMI) and waist circumference correlate with spirometry values in obese individuals.

Methods: We studied 96 non-smokers of both sexes, all suffering from class I or class II obesity and ranging in age from 18 to 75. All participants presented a BMI between 30 kg/m^2 and 40 kg/m^2 and none had a history of morbidity. Spirometry was performed, and waist circumferences were measured.

Results: No significant differences were found between the spirometric values of men with class I or II obesity and those of non-obese men. In obese women, forced vital capacity and forced expiratory volume in one second (FEV_1) were significantly lower than in women who were not obese. Obese individuals of both sexes presented significantly lower expiratory reserve volume (ERV) than did non-obese individuals. Although inspiratory capacity was greater in obese men and women, the difference was significant only for the men. In obese men, there was a significant negative correlation, not seen in the women, between waist circumference and FEV_1.

Conclusion: Pulmonary function is altered in women suffering from class I or II obesity. In obese men, although pulmonary function is unaffected by BMI, we observed a significant negative correlation between BMI and ERV. We can conclude that pulmonary function is influenced by waist circumference in men suffering from class I or II obesity.

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INTRODUCTION

Obesity can affect the thorax, diaphragm, and abdominal muscles and, due to increased respiratory effort and impairment of the gas transport system, can result in altered respiratory function even if the lungs are normal[1-6]. Obesity may also cause hypertonicity in the abdominal muscles, impairing the diaphragmatic activity-dependent respiratory function[7]. Studies of obese individuals not diagnosed with other diseases have suggested that pulmonary and chest wall compliance was reduced due to fat deposition in the chest and abdomen, thereby causing elastic retraction and reduced distensibility of extrapulmonary structures[1,8]. Obesity can be classified using the formula weight/height2 to calculate body mass index (BMI)[9]. We designated the ranges 30-34.9 kg/m2, 35-39.9 kg/m2 and ≥ 40 kg/m2 as obesity classes I, II and III, respectively.

It has been well-established that class III obesity may alter spirometric values by impairing diaphragmatic and thoracic muscle dynamics. However, these changes vary considerably in individuals with class I or II obesity and require specific evaluation[10]. The objective of this study was to determine the effects of class I and II obesity on pulmonary function.

METHODS

We studied 96 adults of both sexes from 30 January to 30 July 2001. Subjects were divided into 4 groups: 24 males suffering from class I or II obesity; 24 non-obese males; 24 females suffering from class I or II obesity; and 24 non-obese females. These individuals were enrolled in the study only after giving written informed consent. The *Santa Casa de São Paulo* Ethics Research Committee approved the study protocols. Data collected included name, age, gender, race, height, weight, *Santa Casa* identification number, date, BMI, waist circumference and spirometric values, as well as information (gathered by questionnaire) regarding previous morbidities.

Considering the objectives of the study, we adopted the following inclusion criteria: class I or class II obesity, 18 to 57 years of age, BMI from 30-40 kg/m^2, sedentary and no history of morbidity. Exclusion criteria were smoking or history of smoking, diabetes mellitus, sinus disease, systemic arterial hypertension, pulmonary disease, cardiac disease, psychiatric disorders, blood disease and other systemic diseases. Other exclusion criteria were prior use of bronchodilators and bronchoconstrictors, recent hospitalizations and surgery within the last 6 months. Control groups were composed of non-obese non-smokers of both genders, 18 to 57 years of age, with BMI from 18.5-24.9 kg/m^2 and no history of morbidities. For all subjects, waist circumference was measured from the midpoint between the last costal arch and the anterior superior iliac crest using a metric tape measure.

Participants were weighed without shoes and with minimal clothing. Height was measured with the aid of an anthropometer coupled to the scale. The weight/height^2 formula was used to calculate BMI.

Pulmonary function tests were carried out in the *Santa Casa de São Paulo* Pulmonary Function Testing Laboratory. The same team of assistants conducted all tests. We used a 1998 Koko spirometer (Pulmonary Data Service, Louisville, CO, USA) with a pneumotachograph coupled to a computer. Spirometric evaluations were carried out in the morning, and volume-time and flow-volume curves were determined.

Individuals were instructed to rest for 5 to 10 minutes prior to the test. The procedures to be carried out were carefully explained to the participants, with an emphasis on maximum inhalation followed by maximum exhalation (sustained until asked to inhale again) and on not allowing air to leak from around the mouthpiece (the technician demonstrated the procedure using a small tube). The area in which the tests were carried out was quiet and private, and temperature and humidity were maintained at constant levels. Tests were carried out between 8:00 and 12:00 am in order to avoid circadian influences. Participants were asked to remain seated during the tests and to wear a nose clip. No bronchodilation tests were carried out. The objective of the spirometry was to measure lung volume and lung capacity, considering values similar to those established by Crapo et al. in 1982 as normal[10]. Spirometric determination of the volume-time curve was carried out in accordance with the criteria established by the American Thoracic Society (1987-1995)[11], and the best of

TABLE 1
Anthropometric data, body mass index, waist circumference, and spirometry values in males with class I or class II obesity and in non-obese males

VARIABLE	OBESE	NON-OBESE	*p*
n	24	24	
Age (years)	31.4 ± 10.5	29.5 ± 7.27	NS
Height (m)	1.78 ± 0.07	1.76 ± 0.07	NS
Weight (kg)	106.5 ± 9.29	69.9 ± 8.11	< 0.05
BMI (kg/m^2)	33.7 ± 2.55	22.3 ± 1.89	< 0.05
WC (cm)	114.3 ± 5.88	81.7 ± 5.23	< 0.05
FVC (L)	5.08 ± 0.59	5.14 ± 0.69	NS
FEV_1 (L)	4.17 ± 0.50	4.39 ± 0.50	NS
FEV_1/FVC	99.6 ± 6.51	103 ± 4.54	NS
$FEF_{25-75\%}$ (L/s)	4.18 ± 1.30	4.83 ± 0.88	NS
ERV (L)	1.16 ± 0.44	1.90 ± 0.34	< 0.05
% ERV	69.4 ± 23.8	119 ± 21.9	< 0.05
IC (L)	3.99 ± 0.39	3.30 ± 0.64	< 0.05
% IC	110 ± 10	92.5 ± 14.1	< 0.05

BMI: body mass index; WC: waist circumference; FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; FEV_1/FVC: ratio of FEV_1 to FVC; $FEF_{25-75\%}$: forced expiratory flow between 25% and 75% of FVC; ERV: expiratory reserve volume; % ERV: percentage of predicted ERV values; IC: inspiratory capacity; % IC: percentage of predicted values for inspiratory capacity; NS: not significant

the 3 acceptable curves was selected. From this curve, we calculated forced vital capacity (FVC), forced expiratory flow in one second (FEV_1), and forced expiratory flow between 25% and 75% of FVC ($FEF_{25-75\%}$), which were analyzed in accordance with the values predicted by Knudson et al. in1983[12]. The same pulmonologist evaluated the results of all pulmonary function tests. Spirometric parameters (absolute and predicted values) were: FVC, FEV_1, FEV_1/FVC ratio, $FEF_{25-75\%}$, expiratory reserve volume (ERV) and inspiratory capacity (IC).

We used the statistical software package SPSS, version 10.0 for Windows. Since the data distribution was normal, the parametric statistical studies employed were Pearson's correlation analysis and non-paired Student's *t*-test. Variables were expressed as means and standard deviations, and *p* values lower than 5% was considered statistically significant.

RESULTS

No significant differences in age or height were found among the various groups studied, indicating that the samples were homogeneous in this respect. However, as expected, weight, BMI and waist circumference were significantly different between obese and non-obese groups (Tables 2 and 3).

There were no significant differences between non-obese and class I and II obese males when we compared FVC, FEV_1 and $FEF_{25-75\%}$ values (Table 2). However, ERV values were significantly lower in obese males and females when compared to those values obtained from non-obese males and females (Tables 2 and 3). In obese males and females, IC values were higher, although the difference was only significant for males (Tables 2 and 3).

Obese females presented significantly lower FVC and FEV_1 values than did non-obese females (Table 3).

In obese males, negative correlations were found between BMI and ERV and between waist circumference and FEV_1 (Table 4). These correlations were less than significant in obese females (Table 5).

Among the individuals suffering from class I or II obesity, no significant alterations were seen in males, although females presented significant decreases in FVC and FEV1 (Tables 1 and 2). This can be explained by the fact that, obesity aside, women have less respiratory muscle strength and therefore produce lower dynamic compression[12,13]. The hyperventilation caused by the effect of progesterone on the bulbar respiratory neurons,

TABLE 2
Anthropometric data, body mass index, waist circumference, and spirometry values in males with class I or class II obesity and in non-obese males

VARIABLE	OBESE	NON-OBESE	*p*
Age (years)	33.5 ± 8.20	34.4 ± 8.16	NS
Height (m)	1.60 ± 0.05	1.61 ± 0.05	NS
Weight (kg)	88.7 ± 8.85	57.3 ± 5.95	< 0.05*
BMI (kg/cm^2)	34.3 ± 2.74	22.0 ± 1.66	< 0.05*
WC (cm)	104 ± 7.40	74.3 ± 4.74	< 0.05*
FVC (L)	3.36 ± 0.56	3.70 ± 0.52	< 0.05*
FEV_1 (L)	2.89 ± 0.48	3.20 ± 0.39	< 0.05*
FEV_1/FVC	100 ± 4.80	101 ± 6.11	NS
$FEF_{25-75\%}$ (L/s)	3.47 ± 0.77	3.89 ± 0.91	NS
ERV (L)	0.71 ± 0.38	1.29 ± 0.36	< 0.05*
% ERV	58.9 ± 29.3	107 ± 35.1	< 0.05*
IC (L)	2.68 ± 0.51	2.44 ± 0.38	NS
% IC	122 ± 21	109 ± 16.2	< 0.05*

BMI: body mass index; WC: waist circumference; FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; FEV_1/FVC: ratio of FEV_1 to FVC; $FEF_{25-75\%}$: forced expiratory flow between 25% and 75% of FVC; ERV: expiratory reserve volume; % ERV: percentage of predicted ERV values; IC: inspiratory capacity; % IC: percentage of predicted values for inspiratory capacity; NS: not significant
*Significance set at $p < 0.05$; variables expressed in means and standard deviations.

airways, and diaphragm may also explain these alterations[14].

Obesity and pregnancy are common causes for reduced FVC since they may interfere with diaphragm movement and chest wall excursion[15]. In 1998, Sahebjami[16] studied pulmonary function in 8 healthy obese males and reported spirometric values similar to those found in the present study, although those authors studied subjects who were in a higher age bracket and presented slightly lower BMIs.

Although abnormalities in the pulmonary function of obese individuals have been described for more than 40 years, there has been considerable variation in the reported degree of alteration, and no definite correlation with body weight or BMI has been shown[17].

There two types of alterations in respiratory function most frequently found in obesity: changes proportional to obesity (reduced ERV and increased diffusion capacity) and alterations exclusive to class III obesity (reduced vital capacity and lower total lung capacity). The reduction in ERV and in functional residual capacity seen in obesity are caused by altered chest wall mechanics, decreased total respiratory compliance, decreased flow frequency, decreased lung volume, reduced residual volume and reduced residual volume/total lung capacity ratio. However, this reduction is not uniform in individuals with similar BMI[17-23].

In a study conducted in 1993, it was also reported that, in pulmonary function tests of obese individuals, ERV (decrease) was the most sensitive parameter and that altered (decreased) ERV becomes more pronounced in parallel with increased obesity (from class I to class II and from class II to class III)[24]. In the present study, we also observed a significant decrease in ERV in both genders, and our data were compatible with those reported in the literature.

Although volume restriction is moderate in obesity, vital capacity is inversely proportional to BMI. In the present study, an inverse, although less than significant, correlation between FVC and BMI was seen in obese males. A positive correlation, also less than significant, was found between FVC and BMI in obese females (Tables 3 and 4). However, in non-obese individuals, vital capacity is directly proportional to BMI. Consequently, the relationship between vital capacity and BMI (or

body weight) in large populations presents an initial increase and a subsequent decrease due to the fact that determination of BMI does not take into account fat distribution within the organism[27]. We detected a directly proportional relationship between FVC and BMI in non-obese males and females (control group) in our study, which confirms the data reported by Lazarus *et al.*[27].

In 1998, Sahebjami and Gartside[22] reported higher percentage of predicted ERV values and lower percentage of predicted IC values than those found in our study. In their study, patients were younger and had lower BMI, which may explain this difference. Their data are similar to those reported in the literature, that is, although class I and II obesity may alter spirometric values, only class III obesity promotes significant impairment of pulmonary function.

In our study, we observed significantly higher IC values in males and females with class I or II obesity than in non-obese individuals. In 1983, Ray et al. also reported similar results[19]. These authors reported that, due to a compensatory increase in IC, vital capacity and total lung capacity were unaltered in class I and II obesity. Increased IC indicates normal pulmonary compliance and the ability of the inspiratory muscles to compensate, at least temporarily, for the fat deposition in the chest and abdominal walls (Tables 2 and 3).

In the present study, no significant correlations were found between BMI and the following spirometric values in either gender: FVC, FEV_1, FEV_1/CVF, and $FEF_{25-75\%}$. The correlation between BMI and ERV, as well as between BMI and percentage of predicted ERV values, was negative and, due to their greater abdominal fat deposition, was only significant in males (Table 3).

In Brazil, spirometric values derived for adults did not relate to weight[13]. In children, although weight contribution was small, there was significant influence[28]. Weight, probably because it reflects an increase in muscle mass, has considerable influence on function values in teenagers[28]. Schoenberg et al. (1978), Dontas et al. (1984) and Chen et al. (1993) reported that increased BMI may result in decreased pulmonary function[29,30,31].

In women, fat deposition is predominantly gynecoid (hips and thighs), whereas it is predominantly abdominal in males. This predominance of abdominal deposition may explain the significant negative correlation between BMI and ERV in males with class I or II obesity.

As a consequence of obesity, especially in those cases presenting abdominal fat deposition, ventilation of the base of the lungs is reduced, especially in those individuals presenting lower ERV values[32].

Various studies have shown altered pulmonary function in individuals with class III obesity. However, there have been few studies involving obese individuals with lower BMI[29,31,33].

TABLE 3

Correlation between body mass index and waist circumference, between body mass index and spirometric values, and between waist circumference and spirometric values in males with class I or class II obesity

Variable pair	Correlation	*p*
BMI vs. WC	positive	NS
BMI vs. Variables (1)	negative	NS
BMI vs. ERV	negative	< 0.05*
BMI vs. % ERV	negative	< 0.05*
WC vs. IC	positive	NS
WC vs. Variables (2)	negative	NS
WC vs. ERV	negative	< 0.05*
WC vs. FEV1	negative	< 0.05*

BMI: body mass index; WC: waist circumference; NS: not significant; ERV: expiratory reserve volume; % ERV: percentage of predicted ERV values; FEV_1: forced expiratory volume in one second; Variables (1): FVC (forced vital capacity), FEV_1, FEV_1/FVC (ratio of FEV_1 to FVC), $FEF_{25-75\%}$ (forced expiratory flow between 25% and 75% of FVC), % ERV; Variables (2): FVC, FEV_1/FVC, $FEF_{25-75\%}$, % ERV
*Significance set at $p < 0.05$; negative sign: the higher the BMI, the lower the value of the variable under study

TABLE 4

Correlation between body mass index and waist circumference, between body mass index and spirometric values, and between waist circumference and spirometric values in females with class I or class II obesity

Variable pair	Correlation	*p*
BMI vs. Variables (1)	negative	NS*
WC vs. Variables (2)	positive	NS
WC vs. ERV and IC	negative	NS*
WC vs. Variables (3)	positive	NS

BMI: body mass index; NS: not significant; ERV: expiratory reserve volume; WC: waist circumference; Variables (1): FVC (forced vital capacity), FEV_1 (forced expiratory volume in one second), FEV_1/FVC (ratio of FEV_1 to FVC), $FEF_{25-75\%}$ (forced expiratory flow between 25% and 75% of FVC); Variables (2): FVC, FEV1/FVC, $FEF_{25-75\%}$, % ERV (percentage of predicted ERV values); Variables (3): FVC, FEV1, FEV1/FVC, $FEF_{25-75\%}$
*Significance set at $p < 0.05$; negative sign: the higher the BMI, the lower the value of the variable under study

In 1978, Schoenberg et al.[29] reported that, due to the related increase in muscle strength, pulmonary function initially increases in parallel with weight gain, although subsequent impairment of chest wall mobility results in reduced pulmonary function. Although several studies have shown that body weight may affect pulmonary function, these data are still in question[29,31,34]. Due to this muscle effect, higher BMI in young individuals may be accompanied by an increase in pulmonary function. In elderly people, however, due to greater fat deposition, increased BMI is associated with decreased pulmonary function. Consequently, the overall impact of BMI on pulmonary function in population studies may be reduced[34]. Most authors agree that weight does not influence spirometric results, or that it does so only in cases of extreme obesity. However, whether weight should be included or not depends on the characteristics of each specific population[29]. In the present study, we observed that increased BMI did not impair pulmonary function in males or females with class I or II obesity.

In obese males, increased waist circumference was accompanied by a significant reduction in FEV_1 and ERV values. This can be explained by the larger mean waist circumference seen in obese males (10 cm larger than in obese females). The correlation between waist circumference and the other spirometric variables was not statistically significant in males or females with class I or II obesity. In 1998, Lean et al. reported a negative correlation between waist circumference and FEV_1/FVC ratio[15]. We observed the same negative correlation in our study, although it was less than significant in obese males. There was positive, although also less than significant, correlation between waist circumference and FEV_1/FVC ratio in females with class I or II obesity. The explanation for this negative correlation has not been well established. However, it is believed that increased waist circumference may have mechanical effects on pulmonary function, and that these effects could be partially explained by impaired movement of the diaphragm and chest wall.

In 1998, in a study on the effects of body mass composition and fat distribution on respiratory function, Lazarus et al.[27] reported that the correlation between FVC and waist circumference was negative in males and positive in females. Although we found similar correlations in the present study, none reached the level of statistical significance.

The isolated effects of obesity unassociated with other diseases must be identified and the analysis of those effects should be stratified so that obesity-related respiratory dysfunction may be evaluated in detail. This aspect is extremely important due to the currently elevated prevalence of obesity and respiratory disease in our society. Although the findings of the present study were inconclusive, they suggest that individuals with class I or II obesity and presenting greater morbidity may be more susceptible to alterations in respiratory function due to lower expiratory reserve. The abnormalities found in the spirometric evaluation of individuals with class I or II obesity must be attributed to respiratory disease and concomitant obesity.

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