

Journal of Respiratory and CardioVascular Physical Therapy

ORIGINAL ARTICLE

EVALUATION OF CHEST WALL KINEMATICS DURING REST AND RESPIRATORY MUSCLE ENDURANCE IN OBESE PATIENTS

ANGELO AUGUSTO PAULA DO NASCIMENTO¹, GUILHERME AUGUSTO DE FREITAS FREGONEZI², SÍLVIA BRILHANTE¹, KAREN LARISSA RODRIGUES SOARES³, VANESSA REGIANE RESQUETI⁴, SELMA SOUSA BRUNO²

¹-Postgraduate Program in Physical Therapy, PneumoCardioVascular Laboratory - Federal University of Rio Grande do Norte, Natal, Rio Grande do Norte, Brasil.

²-Lecturer of Postgraduate Program in Physical Therapy - Federal University of Rio Grande do Norte, Natal, Rio Grande do Norte, Brasil.

³-Undergraduate course in Physiotherapy, Pneumocardiocvascular Physiotherapy Laboratory - Federal University of Rio Grande do Norte, Natal, Rio Grande do Norte, Brazil.

⁴-Department of Physiotherapy, Federal University of Rio Grande do Norte, Natal, Rio Grande do Norte, Brazil. Scholarship DTI-1 CNPq.

Received Month Day, Year; accepted Month Day, Year

KEYWORDS:
Kinematics,
Obesity,
Respiratory
function tests,
Maximal
Voluntary
Ventilations.

Background: Obesity may affect the respiratory system, causing changes in respiratory function and in the pulmonary volumes and flows. **Objectives:** To evaluate the influence of obesity in the movement of thoracoabdominal complex at rest and during maximal voluntary ventilation (MVV), and the contribution between the different compartments of this complex and the volume changes of chest wall between obese and non-obese patients. **Materials and Methods:** We studied 16 patients divided into two groups: the obese group (n = 8) and group non-obese (n = 8). The two groups were homogeneous in terms of spirometric characteristics (FVC mean: 4.97 ± 0.6 L - $92.91 \pm 10.17\%$ predicted, and 4.52 ± 0.6 L - $93.59 \pm 8.05\%$), age 25.6 ± 5.0 and 26.8 ± 4.9 years, BMI 24.93 ± 3.0 and 39.18 ± 4.3 kg/m² in non-obese and obese respectively. All subjects performed breathing calm and slow and maneuver MVV, during registration for optoelectronic plethysmography. Statistical analysis: we used the unpaired t test and Mann-Whitney. **Results:** Obese individuals had a lower percentage contribution of the rib cage abdominal (RCa) during breathing at rest and VVM. The variation of end expiratory (EELV) and end inspiratory (EILV) lung volumes were lower in obese subjects. It has been found asynchrony and higher distortion between compartments of thoracoabdominal complex in obese subjects when compared to non-obese. **Conclusions:** Central obesity impairs the ventilation lung, reducing to adaptation efforts and increasing the ventilatory work.

Corresponding Author

Selma Sousa Bruno (sbruno@ufrnet.br)

INTRODUCTION

It is well documented that, for the most extreme degrees of adiposity, the excess of adipose tissue around the chest can profoundly affect the respiratory system, causing changes in respiratory mechanics and gas exchanges.¹⁻³ When pulmonary restriction is evidenced, it is attributed to the decrease in diaphragmatic excursion by the increase in abdominal adiposity or weight in the chest wall, leading to a reduction in lung volumes when compared with the predicted values.⁴ The spirometric abnormalities most commonly reported in the obese are a reduction in the expiratory reserve volume (ERV) and the functional residual capacity (FRC), with the latter being exponentially related to the body mass index (BMI)^{5,6}. Such restriction has been observed only in patients with alveolar hypoventilation syndrome, since they are carriers of extreme obesity.¹⁻³

Increases in BMI also progressively increase the prevalence of symptoms, such as the sensation of dyspnea at rest⁷. Besides, the pattern of body fat distribution, even before the BMI alone, can also have a more negative influence on the lung volumes of obese subjects than the BMI alone⁸. Thus, other measures of adiposity such as waist (WC)⁹ and neck (NP)¹⁰ and waist-hip (WHR)⁸ have been taken as important predictors of lung function.

Lung ventilation is functionally the product of two compartments, thoracic and abdominal, working in sync during the respiratory act¹¹. When the displacement between these compartments becomes asynchronous, the thoracoabdominal movement can contribute to changes in ventilation efficiency, increasing respiratory work and maximum oxygen consumption^{12,13}. The study of thoracoabdominal asynchrony has been highlighted in several areas¹⁴⁻¹⁷. Especially in the obese, such asynchrony can reflect in the reduction of the ventilation in the pulmonary base leading to the early closure of the peripheral lung units. The study of thoracoabdominal asynchrony has been especially important in the postoperative period where these abnormalities tend to be exacerbated and in the evaluation of the effectiveness of techniques for increasing lung ventilation¹⁴.

The increase in abdominal volume, among other aspects, can modify the diaphragmatic curvature and its apposition zone and location in the lower region of the rib cage, which compromises the efficiency of this muscle and the overload of others¹⁸. Initial studies have shown divergent results about static muscle strength in obesity^{19,20}. However, there seems to be a consensus regarding the ability to generate high pulmonary flows, translated by the decrease in maximum voluntary ventilation (MVV), which appears to be altered especially in central adiposity^{10,21}.

Although several hypotheses have been attributed to justify the decrease in ventilatory performance in the obese, there still seems to be no complete understanding of the restrictive respiratory disorder observed, especially with regard to the study of thoracoabdominal asynchrony at rest and in high pulmonary flow maneuvers. Thus, the objectives of this study were to evaluate the influence of obesity on the movement of the thoracoabdominal complex during rest and maximum voluntary ventilation, as well as the contribution between the different compartments of this complex (Pulmonary rib cage - RCp), Abdominal rib cage - RCa and Abdominal - Ab) and changes in chest wall volume and between obese and non-obese individuals.

MATERIALS AND METHODS

Subjects

The study is a cross-sectional study, with the collection being carried out from September 2010 to January 2011, with groups of obese and non-obese individuals, carried out at the Pneumocardiopulmonary Physiotherapy Lab of the Federal University of Rio Grande do Norte, by the same evaluator, physiotherapist, trained with the technique, during the night.

The sample was selected by convenience, participating in the study, volunteer men distributed in two groups: obese group with central obesity (WHR = 1.0 ± 0.01); and a group of eutrophic. Inclusion criteria were: obese with BMI between 35 kg/m² and 45 kg/m², all-male, active, able to perform respiratory examinations (spirometry, optoelectronic plethysmography, and MVV), aged between eighteen and forty years, with no history of previous lung disease or chest surgery in the last six months before the study. Present the Forced Vital Capacity (FVC) greater than

80% of the predicted individual and the relationship between the maximum expiratory volume per second and the FVC greater than 80% of the predicted^{22,23}. Patients with neurological deficits were excluded from the sample, those who were unable to perform all the necessary procedures for evaluation, or those who, at any time, requested to withdraw from participating in the study. As a preparatory recommendation, all individuals were informed about not having teas, drinks containing caffeine, soda or using any stimulant less than three hours before the evaluation. Strict physical exercise restriction was also indicated in the twelve hours before the assessment²³. All subjects, before being admitted to the study, signed the Free and Informed Consent Form. The study was approved by the Research Ethics Committee (CEP) under nº 148/07, of the Federal University of Rio Grande do Norte (UFRN), according to resolution 196/96 of the National Health Council.

Physical activity level and BMI

Initially, individuals were assessed for anthropometry and level of physical activity, measured BMI, WC, CQ, and WHR according to the standard previously described^{24,25} and thus determined the degree of obesity and the pattern of distribution of body adiposity.

Spirometry

Spirometry was previously performed on all individuals to characterize a healthy sample from a spirometric point of view. Thus, individuals were included according to the criteria previously defined. The exam was performed according to the acceptance standards previously described²³. The equipment used was the DATOSPIR 120 (SibelMed Barcelona, Spain) coupled to a microcomputer and calibrated daily. The VVM maneuver was performed over a period of ten to fifteen seconds, as previously described²³. All maneuvers were recorded on the spirometer itself, while the pressure behavior of the test was recorded on the Micro Medical Software software (Spida 5, Puma).

Thoracoabdominal complex kinematics

The individuals were previously instructed on the instrumentation, and the ventilatory pattern to be adopted in each capture maneuver. The collection of pulmonary ventilation variables was collected in two moments: at rest with the VC maneuver and then (time) with the VVM maneuver.

For the evaluation of the thoracoabdominal complex kinematics, we used the optoelectronic plethysmograph (OEP, BTS, Milan, Italy), whose motion analysis was captured by six cameras arranged along the room (3 positioned before the subject and 3 positioned later).

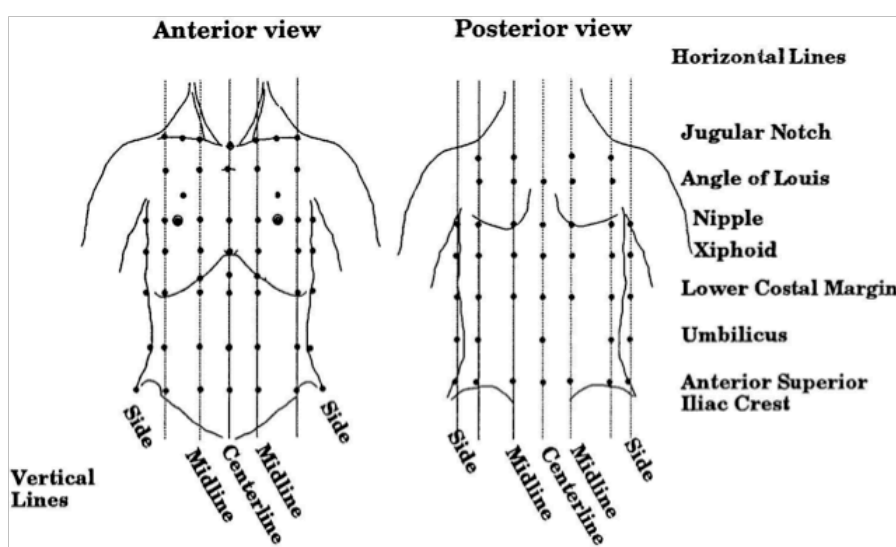


Figure 1 - General arrangement of markers. Anterior and Posterior View.

We used a model of protocol made with 89 reflective infrared markers, fixed on the subject's chest using bi-adhesive hypoallergenic tape over the subjects' skin, positioned in a grid system consisting of seven horizontal lines arranged circumferentially between the level of the clavicle and the anterior superior iliac spine. Along these lines, the markers were positioned anteriorly (42), posteriorly (37) and laterally (10) in five columns and an additional bilateral column in the middle axillary line^{26,27}. The subjects were placed in a seated position on a backless bench, centered on a system of six previously calibrated cameras. An image processor detected the reflection of the infrared at the points, allowing the specific software to design a model in three dimensions, linking the markers in various triangulations, which, based on Gauss' theorem, provided continuous measurements of the volumes of the entire trunk (Chest Wall), in addition to compartmentalization of the trunk into three components: an upper one, the pulmonary rib cage (Rib cage pulmonary - RCp); one intermediate, the abdominal rib cage (Rib cage abdominal - RCa) and one lower, the abdomen (Abdominal - Ab). Changes in the thoracoabdominal complex were recorded in the OEP Capture26 software. For the measures of thoracoabdominal synchrony, the following measures were taken: changes (Δ) in volume between breathing at rest and VVM maneuver, the percentage of contribution of each chest wall compartment, the phase angle (PhAng - reflects delay in excursions of the chest wall compartments), the inspiratory phase relationship (PhRIB - expresses the percentage of inspiration in which the movement of the rib cage and abdomen are in the opposite direction), the expiratory phase relationship (PhREB - expresses the percentage the expiration in which the movement of the rib cage and abdomen are in the opposite direction) and the total respiratory phase relationship (PhRTB - expresses the percentage of the breathing cycle in which the movement of the rib cage and abdomen are in the opposite direction).

Statistical analysis

Statistical analysis was performed using the GraphPad Prism 4.0 for Windows software. For all data, we applied the Shapiro-Wilk test to verify the normality of the distribution. To verify the difference in means between the

obese and eutrophic groups, with normal distribution, we applied the unpaired t test. For those who were not parametric, we used the Mann Whitney test.

RESULTS

Twenty individuals were analyzed, 12 obese and 8 eutrophics. Of these, 4 obese were excluded due to technical problems in the mathematical models, which did not allow the analysis of volumes. Thus, 8 individuals were studied in the obese group and 8 in the eutrophic group. The individuals in both groups were homogeneous in terms of spirometric characteristics (mean FVC: 4.96 L - 92% of predicted, and 4.52 L - 93%), age 25.6 and 26.8 years in eutrophic and obese individuals, respectively. As expected, there was a significant difference concerning the groups' anthropometric measurements. The mean BMI of non-obese individuals was 24.9 and 39.2 kg / m² in obese individuals ($p < 0.0001$). The general baseline characteristics of the other anthropometric and spirometric measurements of the individuals are shown in table 1.

Table 2 presents the results of the percentage of contributions to the ventilation of the chest wall compartments between the groups. During tidal volume at rest, it was observed that Ab contributes more to the result of ventilation in both groups. In this maneuver, the contribution of compartment Ab is followed by RCp and RCa, which contributes less to ventilation than RCp and Ab. In this respect, the contribution of RCa was lower ($p = 0.01$) in obese individuals for the same compartment than in non-obese individuals.

During the VVM maneuver, non-obese individuals modified the compartment contribution pattern, with RCa contributing in second place to pulmonary ventilation. About non-obese patients, the pattern of contribution to pulmonary ventilation in the RCa compartment is much lower compared to non-obese patients ($p = 0.07$).

Volume variations between obese and eutrophic subjects were obtained by comparing breathing at rest and the MVV maneuver between the two groups. A similar variation (Δ) was observed in the tidal volume of the chest wall (Chest Wall - Cw) between the two groups studied and a significant percentage difference and in liters in the variations in expiratory lung volumes ($p = 0.02$, for the percentage, $ep = 0.003$, for liters) and final inspiratory (p

<0.0001, for the percentage, and $p = 0.008$, for liters) (EELV and EILV), as shown in table 3.

Table 4 shows the comparison of asynchrony and distortion variables between obese and eutrophic individuals. The phase angle (PhAng) between the pulmonary ribcage (RCp) and the abdominal ribcage (RCa) of the obese was greater than that of the eutrophic ($p = 0.03$). The inspiratory phase (PhRIB) and total respiratory phase (PhRTB) relationships were higher in obese subjects, comparing all compartments of the chest wall (PhRIB AB x RC, $p = 0.03$; RCa x RCp, $p = 0.003$; AB x RCa, $p = 0.006$; PhRTB AB x RC, $p = 0.03$; RCa x RCp, $p = 0.005$; AB x RCa, $p = 0.009$) while the expiratory phase relationship (PhREB) did not show statistical significance only between the

abdominal compartment and the rib cage (PhREB RCa x RCp, $p = 0.008$; AB x RCa, $p = 0.02$).

DISCUSSION

The present study was conducted to assess lung volumes and the contribution of thoracoabdominal complex compartments to obesity in breathing at rest and during the MVV maneuver. A second objective was to investigate the presence of asynchrony and distortion between the thoracoabdominal compartments, in the obese and eutrophic control subjects. The percentage contribution of the rib cage during ventilation was lower in breathing at rest, worsening during the MVV maneuver. The variations of EILV and EELV were also lower in obese subjects, when compared to the control group. There was distortion in PhAng RCaxRCp and distortions and asynchrony in the analysis of PhRIB, PhREB and PhRTB, for all compartments. To our knowledge, this is the first time that these results have been presented.

The proper functioning of the respiratory system requires that the lungs and chest wall have normal compliance and that the muscles have the necessary strength and tone, with the diaphragm remaining aciform, making it possible, through its contraction, to increase the volume of the chest. thoracic¹⁸. In obese individuals, the diaphragm muscle is located more superiorly in the abdomen compared to individuals without abdominal obesity. Our obese group had a predominantly higher body fat distribution pattern (WHR = 1.1). Although the abdominal distribution was measured indirectly, we believe, similar to what is pointed out in other studies²⁸⁻³⁰, that the excessive deposition of adipose tissue around the chest and abdominal wall can change the length-tension curve of the diaphragm, these facts may support the decrease in the contribution of RCa at rest.

In obese individuals, the diaphragm muscle is located in a superior position. These facts can support the lower contribution of RCa (27.12 ± 5.63 , $p < 0.010$) at rest. Study participants had an android pattern of adiposity distribution, suggesting changes in diaphragmatic apposition and possible impairment of their function due to changes in the length-tension curve.

Table 1- Anthropometric data, spirometric variables and physical activity level of the participants.

	Eutrophics	Obese
Sample	8	8
Anthropometric Variables		
Age (years)	25,63 ± 5,07	26,88 ± 4,19
Weight (kg)	76,20 ± 12,43	117,80 ± 8,42*
Height (cm)	175,3 ± 4,89	173,5 ± 8,00
BMI (kg/m ²)	24,93 ± 3,02	39,18 ± 4,36*
Spirometric Variables		
FVC(L)	4,97 ± 0,60	4,52 ± 0,63
FVC%	92,91 ± 10,17	93,59 ± 8,05
FEV ₁ (L)	4,21 ± 0,53	3,87 ± 0,55
FEV ₁ %	94,55 ± 9,40	94,02 ± 10,62
FEV ₁ /FVC	82,27 ± 10,25	84,90 ± 3,63
FEV ₁ /FVC%	97,68 ± 11,99	84,64 ± 2,02*
MVV (L/min)	159,3 ± 21,44	155,1 ± 17,44
MVV%	87,84 ± 16,01	91,33 ± 9,20
Physical Activity Level		
HAP (AAS)	87,13 ± 5,22	80,2 ± 5,4

FVC = forced vital capacity, FVC% = percentage of predicted values for FVC, FEV₁ = forced expiratory volume in one second, FEV₁% = percentage of predicted values for FEV₁, FEV₁ / FVC = ratio between FVC and FEV₁, FEV₁ / FVC% = percentage of predicted values for FEV₁ / FVC, MVV = maximum voluntary ventilation, MVV% = percentage of predicted values for MVV, AAS = adjusted activity score by the Human Activity Profile. *Significant difference ($p < 0,05$)

Table 2 - Percentage contribution of chest wall compartments in ventilation between obese and eutrophic subjects during QB and MVV maneuver.

Variables	Eutrophics	Obese
RC _{POB} %	35,26 ± 7,15	34,80 ± 9,98
RC _{AOB} %	27,12 ± 5,63	19,36 ± 4,84*
Ab _{OB} %	37,62 ± 8,56	43,14 ± 12,55
RC _{PMVV} %	26,08 ± 4,13	34,25 ± 11,33
RC _{AMVV} %	38,74 ± 12,95	19,35 ± 11,40*
Ab _{MVV} %	42,29 ± 11,13	46,40 ± 19,46

RC_{POB} % = percentage of contribution of pulmonary rib cage to ventilation during tidal volume, RC_{AOB} % = percentage of contribution of abdominal rib cage, Ab_{OB} % = percentage of contribution of abdomen, RC_{PMVV} % = percentage of pulmonary rib cage contribution to ventilation during maximal voluntary ventilation, RC_{AMVV} % = percentage of abdominal rib cage contribution, Ab_{MVV} % = percentage of abdomen contribution. *Significant difference ($p < 0,05$)

The VVM maneuver during spirometry was similar between groups. This finding is supported by the fact that MVV behaves, in relation to BMI, increasing proportionally in non-obese subjects and presenting an inverse relationship from BMI above 45kg/m²³¹. Although our obese patients have BMI slightly below these values, in the ventilation analysis performed with the POE, the discrepancy in the contribution of the thoraco-pulmonary compartments between the groups was greater during this test. The MVV maneuver requires an increase in respiratory rate, so the diaphragm's relaxation time would be further reduced, due to the hyperdistension caused by central fat. We can attribute the absence of significant changes in the contribution of the RCp and the Ab to the fact that the RCp receives support from the accessory musculature to compensate for the overload in this compartment, while the Ab would be compensated due to being the part of the thoracoabdominal complex more flexible.

We found no differences in $\Delta V_{C_{CW}}$ between eutrophic and obese individuals. A study carried out with eight obese and six eutrophic individuals found that these first compensated the tidal volume with an increase in respiratory rate and with shallow breaths³². However, $\Delta EELV_{CW}$ and $\Delta EILV_{CW}$ were significantly lower in the obese subjects in our study. EELV regulation is critically important in exercise, with activation of expiratory muscles, optimizing the diaphragmatic length for the development of strength and accumulating elastic energy that will be used to assist the next inspiration. Also, this

Table 3 - Chest wall volume variations between obese and eutrophic subjects, comparing resting breathing and MVV maneuver between the two groups.

Variables	Eutrophics	Obese
$\Delta\% V_{T_{CW}}$	53,74 ± 13,60	53,76 ± 6,54
$\Delta V_{T_{CW}}$ (L)	0,87 ± 0,30	1,07 ± 0,14
$\Delta\% EEV_{CW}$	3,18 ± 1,79	0,59 ± 0,98*
ΔEEV_{CW} (L)	0,74 ± 0,40	0,23 ± 0,40*
$\Delta\% EIV_{CW}$	6,33 ± 1,78	1,98 ± 1,44*
ΔEIV_{CW} (L)	1,60 ± 0,42	0,83 ± 0,56*

$\Delta V_{T_{CW}}$ (L) = Variation in liters of tidal volume in the chest wall between resting breathing and maximum voluntary ventilation, ΔEEV_{CW} (L) = Variation in liters of end-expiratory volume in the chest wall between resting breathing and maximal voluntary ventilation, ΔEIV_{CW} (L) = Variation, in liters, of the end-inspiratory volume in the chest wall between resting breathing and the maximum voluntary ventilation. *Significant difference ($p < 0,05$)

phenomenon helps to maintain lung volumes operating within the linear portion of the pressure-volume ratio, decreasing the inspiratory elastic load and keeping the EILV below the CPT and the inspiratory flow within 75% of the maximum available flow³³. Despite not being characterized as an exercise, VVM implies an additional burden to the ventilatory act. In a study evaluating 20 healthy subjects, with normal spirometry, demonstrated that the load generated by this maneuver at the inspiratory peak in men was 34.7 (± 5.3) cmH₂O, and at the expiratory peak, it was 33,8 (± 5.9) cmH₂O³⁴. Another recent article carried out with nine obese women in grade II and eight eutrophic, in a full-body plethysmograph, analyzed at rest and during exercise, demonstrated that the EELV of obese subjects is decreased at rest and declines even more during exercise. EILV, at rest, was higher in obese women, but not during exercise³⁵. Similar evidence was found in a survey of 10 men with grade II obesity and nine eutrophic men, investigated at rest (sitting and supine) and at exercise, using the same plethysmographic methodology and measuring esophageal and gastric pressures. The results showed that grade II obesity resulted in a reduction in VEE at rest and changes in respiratory mechanics³⁶. It is speculated that the reduction in VEE is due to expiratory abdominal forces in the diaphragm, with an increase in gastric pressure related to the distribution of abdominal adiposity^{28,35,8}. Our findings are supported in the literature and suggest a low adaptation of obese individuals to effort, with impairment in the mobilization of volumes.

As for thoracoabdominal asynchrony, studies in obese individuals are still scarce. In a recent study, a group of thirty obese individuals undergoing reductive gastroplasty, aged 32.37 (\pm 8.54) years and a BMI of 42.72 (\pm 4.10) kg / m² and a control group of thirty individuals non-obese patients aged 30.60 (\pm 7.76) years and a BMI of 21.99 (\pm 2.22) kg/m², were assessed through respiratory plethysmography by electrical inductance. Obese people showed a reduction in the labor breathing index (LBI), after six months of surgery, but the PhAng became similar to the preoperative³⁷. A recent publication evaluated the breathing pattern and the thoracoabdominal movement, using the same technique as the previous study, during breathing exercises in 24 patients with obesity levels II and III, of both genders, mean age of 37 (\pm 11) years and BMI 44 (\pm 3) kg / m², on the second postoperative day of gastroplasty. PhAng, PhRIB and LBI were analyzed at rest and during diaphragmatic exercise (DE), flow-oriented incentive spirometry (FIS) and volume-oriented incentive spirometry (VIS). There was a significant increase in PhRIB during ED and FIS, as well as PhAng during FIS and VIS, concerning the respective baseline values, with no significant differences between the three exercises¹⁴.

In the present study, for asynchrony and distortion, obese and eutrophic subjects were compared only at rest and we found statistical significance for the PhAng RCaxRCp, which characterizes distortion between these compartments. It is possible that this finding is correlated with the reduced contribution of RCa to ventilation and this distortion is justified by the fact that RCp presents itself as the compartment of less complacency and the diaphragm muscle has its excursion limited by visceral fat. PhRIB and PhRTB, among all studied compartments, showed significantly higher in obese subjects. PhREB did not present asynchrony only between the abdomen and the rib cage. These findings indicate that central obesity has repercussions throughout the respiratory cycle, in the form of asynchrony and distortion. These data suggest that, although the subjects did not present restrictive disorders from the spirometric point of view, they already had important changes in lung volumes and also in respiratory mechanics, contributing to the increase in ventilation work and characterizing yet another dysfunction related to obesity in the respiratory system .

Table 4 - Comparison of asynchrony and distortion variables between obese and eutrophic individuals.

Variables	Eutrophics	Obese
PhAng ABxRC	7,26 \pm 3,45	10,78 \pm 7,96
PhAng RCaxRCp	5,27 \pm 3,30	15,50 \pm 11,84*
PhAng ABxRCa	6,71 \pm 3,51	12,56 \pm 7,11
PhRIB AB x RC	7,98 \pm 3,23	15,16 \pm 7,82*
PhRIB RCa x RCp	7,49 \pm 3,92	20,64 \pm 9,75*
PhRIB AB x RCa	6,80 \pm 2,60	15,03 \pm 6,79*
PhREB AB x RC	5,55 \pm 4,20	11,75 \pm 7,09
PhREB RCa x RCp	5,34 \pm 3,65	17,87 \pm 10,76*
PhREB AB x RCa	4,85 \pm 4,05	11,39 \pm 5,89*
PhRTB AB x RC	6,77 \pm 3,82	13,30 \pm 6,92*
PhRTB RCa x RCp	6,39 \pm 3,65	18,54 \pm 9,53*
PhRTB AB x RCa	5,89 \pm 3,36	13,23 \pm 5,94*

PhAng ABxRC = phase angle, given in degrees, between the ribcage and the abdomen, PhAng RCaxRCp = phase angle between the pulmonary rib cage and the abdominal rib cage, PhAng ABxRCa = phase angle between the abdominal rib cage and the abdomen, PhRIB = inspiratory phase ratio, given in percentage, PhREB = expiratory phase ratio, given as a percentage, PhRTB = total respiratory phase ratio, given as a percentage. *Significant difference ($p < 0,05$)

The present study has some potential limitations, such as the number of subjects studied. However, the presence of a control group matched for age and respiratory function contributes to the results found to be important to the respiratory mechanics of the obese. Besides, the work opens up the prospect of future studies indicating that it is especially useful in the study of pulmonary mechanics of obesity, since thoracoabdominal asynchrony is one of the elements that probably tends to exacerbate itself mainly in postoperative conditions of the upper floor of the patient abdomen.

In summary, central obesity negatively interferes with the RCa's contribution to ventilation, with reduced EELV and EILV variations, reducing adaptation to efforts. The thoracoabdominal movement, at rest, is altered, due to the distribution of adiposity, with an increase in ventilatory work, compared to eutrophic subjects.

REFERENCES

1. Shimura R, Tatsumi K, Nakamura A, Kasahara Y, Tanabe N, Takaguchi Y, et al. Fat accumulation, leptin, and hypercapnia in obstructive sleep apnea-hypopnea syndrome. *Chest*. 2005; p. 543-9.
2. Berger KI, Ayappa I, Chaturvedi A, Marfatia A, Sorkin I, Barry RRT, et al. Obesity-hypoventilation syndrome as a spectrum of respiratory disturbances during sleep. *Chest*. 2001; p. 1231-8.
3. Phipps PR, Sarritt E, Catterson I, Grunstein RR. Association of serum leptin with hypoventilation in human obesity. *Thorax*. 2002; p. 75-6.
4. Hakala K, Mustajoki P, Aittomäki J. Effect of weight loss and body position on pulmonary function and gas exchange abnormalities in morbid obesity. *Int. J. Obes Relat Metab Disord*. 1995; p. 343-346.
5. O'Donnell P, Holguin F, Dixon AE. Pulmonary physiology and pathophysiology in obesity. *J Appl Physiol*. 2010; p. 197-198.
6. Ceylan E, Comlekci A, Akkoclu A, Ceylan C, Ergor G, Yesil S. The effects of body fat distribution on pulmonary function tests in the overweight and obese. *South Med J*. 2009 Jan; p. 30-5.
7. Sahebajami H. Dyspnea in obese healthy men. *Chest*. 1998; p. 1373-7.
8. Lazarus R, Sparrow D, Weiss ST. Effects of obesity and fat distribution on ventilatory function. *Chest*. 1997; p. 891-8.
9. Lean MEJ, Seidell JC. Impairment of health and quality of life in people with large waist circumference. *Lancet*. 1998; p. 853-6.
10. Gonçalves MJ, Lago STS, Godoy EP, Fregonezi GAF, Bruno SS. Influence of neck circumference on respiratory endurance and muscle strength in the morbidly obese. *Obes Surg*. 2010 february.
11. Sackner MA, Gonzalez H, Rodriguez M, Belsito A, Sackner DR, Grenvik S. Assessment of asynchronous and paradoxical motion between rib cage and abdomen in normal subjects and in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis*. 1984; p. 588-593.
12. Kiciman NM, et al. Thoracoabdominal motion in newborns during ventilation delivered by endotracheal tube or nasal prongs. *Pediatr Pulmonol*. 2000; p. 175-181.
13. Rabler B, Kohl J. Analysis of coordination between breathing and walking rhythms in humans. *Respiration Physiology*. 1996; p. 317-327.
14. Tomich GM, França DC, Diniz MTC, Britto RR, Sampaio RF, Parreira VF. Efeitos de exercícios respiratórios sobre o padrão respiratório e movimento toracoabdominal após gastroplastia. *J Bras Pneumol*. 2010; p. 197-204.
15. Aliverti A, Quaranta M, Chakrabarti B, Albuquerque ALP, Calverley PM. Paradoxical movement of the lower ribcage at rest and during exercise in COPD. *European respiratory journal*. 2009; p. 46-90.
16. Tomich GM, França DC, Diório ACM, Sampaio RF, Parreira VF. Breathing pattern, thoracoabdominal motion and muscular activity during three breathing exercises. *Brazilian Journal of Medical and Biological Research*. 2007; p. 1409-1417.
17. Allen LJ, Wolfson RM, McDowell K, Shaffer HT. Thoracoabdominal Asynchrony in Infants with Airflow Obstruction. *Am Rev Respir Disease*. 1990; p. 337-342.
18. Rasslan Zea. Função Pulmonar e Obesidade. *Rev Bras Clin Med*. 2009; p. 39-39.
19. Sarikaya S, Cimen OB, Gokcay Y, Erdem R. Pulmonary function tests, respiratory muscle strength and endurance of persons with obesity. *The Endocrinologist*. 2003 april; p. 136-141.
20. Castello V, Simões RP, Bassi D, Mendes RG, Borghi-Silva A. Força muscular respiratória é marcadamente reduzida em mulheres obesas mórbidas. *Arq Med ABC*. 2007; p. 74-77.
21. Costa D, Barbalho MC, Miguel GPS, Forti EMP, Azevedo JLMC. The impact of obesity on pulmonary function in adult women. *Clinics*. 2008; p. 719-24.
22. World Health Organization. Fact sheet: obesity and overweight. [Online]; 2010 [cited 2010 Agosto 31]. Available from: <http://www.who.int/mediacentre/factsheets/fs311/en/>.
23. Sociedade Brasileira de Pneumologia e Fisiologia. Diretrizes para Testes de função pulmonar. *J Pneumol*. 2002; p. S207-21.
24. Souza AC, Magalhães LdC, Teixeira-Salmela LF. Adaptação transcultural e análise das propriedades psicométricas da versão brasileira do Perfil de Atividade Humana. *Cad Saúde Pública*. 2006 dez; p. 2623-2636.

25. World Health Organization. Obesity: preventing and managing the global epidemic. Geneva;; 2000.
26. Aliverti A, Pedotti A. Opto-Electronic Plethysmography. *Monardi Arch Chest Dis.* 2003: p. 12-16.
27. Cala SJ, Kenyon CM, Ferrigno G, Carnevali P, Aliverti A, Pedotti A, et al. Chest wall and lung volume estimation by optical reflectance motion analysis. *J Appl Physio.* 1996: p. 2680-2689.
28. Collins LC, Hoberty PD, Walker JF, Fletch EC, Peiris AN. The effect of body fat distribution on pulmonary function tests. *Chest.* 1995: p. 1298-302.
29. Parameswaran K, Todd DC, Soth M. Altered respiratory physiology in obesity. *Can Respir J.* 2006 May: p. 203-210.
30. Chlif M, Keochkerian D, Choquet D, Vaidie A, Ahmaidi S. Effects of obesity on breathing patten, ventilatory neural drive and mechanics. *Respiratory Physiology & Neurobiology.* 2009: p. 198-202.
31. Ladosky W, Botelho MAM, Albuquerque Jr JP. Chest mechanics in morbidly obeses non-hipoventilated patients. *Resp Med.* 2001: p. 281-6.
32. Pankow W, Podszus T, Gutheil T, Penzel T, Peter JH, Von Wichert P. Expiratory flow limitation and intrinsic positive end-expiratory pressure in obesity. *Journal of Apllied Physiology.* 1998 october: p. 1236-1243.
33. Neder JA, Nery LE. Teste de Exercício Cardiopulmonar. *J pneumol.* 2002 outubro: p. s166-s100.
34. Severino FG. Avaliação muscular respiratória: adaptação de do manovacuômetro nacional para avaliação da pressão inspiratória nasal e nível de intensidade da ventilação voluntária máxima em sujeitos saudáveis. 2010. Dissertação de Mestrado.
35. Babb TG, DeLorey DS, Wyrick BL, Gardner PP. Mild obesity does not limit change in end-expiratory lung volume during cycling in young women. *J Appl Physiol.* 2002 june: p. 2483-2490.
36. DeLorey DS, Wyrick BL, Babb TG. Mild-to-moderate obesity: implications for respiratory mechanics at rest and during exercise in youg men. *International Journal of Obesity.* 2005: p. 1039-1047.
37. Matos CMdP. Parâmetros respiratórios e qualidade de vida de indivíduos obesos, antes e após gastroplastia redutora: um estudo longitudinal. 2007. Dissertação de Mestrado.