Journal of
Obesity & Weight Loss Therapy

Herrejón et al., J Obes Weight Loss Ther 2016, 6:4
DOI: 10.4172/2165-7904.1000318

Research Article Open Access

Pulmonary Function in Morbid Obesity: Influence of Sex and Body Distribution

Alberto Herrejón*, Ignacio Inchaurraga and Julio Palop

Department of Pneumology, Dr. Peset University Hospital, Valencia, Spain

***Corresponding author:** Herrejón A, Department of Pneumology, Dr Peset University Hospital, Avda Gaspar Aguilar, 9046017-Valencia, Spain, Tel: 34961622438; E-mail: herrejon_alb@gva.es

Received date: July 26, 2016; **Accepted date:** August 26, 2016; **Published date:** August 29, 2016

Abstract

Objective: To describe the respiratory functional alterations that occur in patients with morbid obesity (MO) and the possible influence of gender and distribution of body fat, measured as an index waist/hip ratio (wai/hip).

Method: Prospective study in 2 years of anthropometric and functional parameters breathing in MO, determining age, body mass index (BMI), waist, hip, wai/hip index, blood gas values, flow-volume curve, plethysmography, diffusion of CO and maximum inspiratory (MIP) and expiratory (PEM) pressures. The results between men and women and the relationship between respiratory parameters and wai/hip index are compared.

Results: We studied 171 patients, 80 men and 91 women, with 44 ± 12 years, weighing 130 ± 22 kg, with BMI of 48 ± 6 Kg/m^2 and wai/hip index of 0.989-0.097 ± 48. There is a decrease in lung function parameters in the MO, showing men MIP worse, but better MEP, than women. Hypoxemia is common (55%), related to an older and worse flows and static volumes. Hypercapnia is 15% more common in men, and is related to the decrease in expiratory flows and increased residual volume. Wai/hip index does not correlate with respiratory parameters in the MO.

Conclusions: Morbid obesity affects respiratory blood gas and has functional alterations. There are differences between men and women in the presence of hypercapnia and maximal respiratory pressures, without influencing the type of obesity messured with the wai/hip index.

Keywords: Morbid obesity; Lung function; Type of obesity

Introduction

MO is a common clinical situation defined by a BMI ≥ 40 kg/m^2. Two types of obesity are recognized, android or central and peripheral or ginoide obesity [1]. Central obesity is the one with rate of wai/hip ratio>1 in men and >0.9 in women [2]. It is associated with increased cardiovascular risk such as hypertension, diabetes or dyslipidemia and also increases the risk of two major respiratory problems: Obesity hypoventilation syndrome and sleep obstructive apneas syndrome [3]. Obesity, in terms of the distribution of body fat, has been linked to alterations in the pulmonary function [4,5].

Respiratory function of patients with MO is poorly studied, usually in limited samples. Two patterns of respiratory functional impairment in the OM are described. In the first pattern functional impairment, which is proportional to the degree of obesity is increasing diffusion of carbon monoxide (DLCO) and DLCO/alveolar ventilation (KCO) relationship, with reduced expiratory reserve volume (ERV). The second pattern, typical of most obese patients, is characterized by a decrease in vital capacity (VC), total lung capacity (TLC) and maximum ventilation voluntary [6]. However, it has not assessed the possible influence of sex and type of obesity on pulmonary function of patients with MO.

The aim of our work is the study of the parameters of conventional lung function in a large group of patients with OM and its relationship to sex and the degree and type of obesity. The importance of the issue is due to the close relationship between lung function and morbid obesity, analyzed a large sample of subjects of both sexes, which condition their clinical situation and are likely to improve with weight reduction.

Material and Methods

Patients

We have studied prospectively patients with MO in a university hospital with an area of 321,361 inhabitants, for 5 years.

Method

We included consecutive patients with MO, excluding those with a history of respiratory disease known. We studied anthropometric and functional parameters.

Registered anthropometric parameters were: age (years), weight (kg), height (cm), BMI (kg/m^2), waist circumference (wai) at the navel (cm) hip circumference (hip) at the level of the greater trochanters (cm) and waist/hip ratio (wai/hip) index, both perimeters made in raising the standing position, if necessary, the abdominal fat mass.

The respiratory function parameters studied were: blood gas analysis, curve forced expiratory flows through flow/volume, static lung volumes with body plethysmography, CO diffusing capacity, maximum respiratory muscle pressures, inspiratory (MIP) and expiratory (PEM). Arterial blood gas analysis was performed in supine, determining pH, PO_2 (mmHg), PCO_2 (mmHg) and sat. O_2 (%) with the ABL 300 gas analyzer Radiometer Co. (Copenhagen), according to

the technique described by Flenley [7]. Hypoxemia was considered a PO_2 below 80 mmHg, severe hypoxemia when the PO2 is less than 60 mmHg and hypercapnia if PCO_2 was greater than 45 mmHg. The flow/volume curve was performed using a pneumotachograph system 2800 SensorMedics (SensorMedics Corporation. California 1984), following the european regulations [8] and registering forced vital capacity (FVC) (L), forced expiratory volume in one second (FEV1) (L/sec) and FEV1/FVC. Static volumes were determined by body plethysmography (Body Box 2800 SensorMedics), measuring total lung capacity (TLC) (L), residual volume (RV) (L) and functional residual capacity (FRC) (L). The diffusion of CO was obtained by the method of breathing unique with the team SensorMedics 2100, following the recommendations of the ATS [9], analyzing the DLCO (mmol/kPa.min) and KCO (mmol/kPa.min/L). Spirometric, plethysmographic and diffusion of CO values are expressed as percentages of the theoretical values, as CECA tables [10]. Maximal respiratory pressures were determined in mouth, nasal occlusion using a portable digital manometer (MicroMPM of SensorMedics. Brethoven Netherland) according to the method of Black and Hyatt [11]. PEM was measured at the level of TLC in cm H_2O, hands on her cheeks to ignore the effect of the buccinator and PIM (expressed in absolute values) in cm H_2O from RV, with a leak of a 1 mm diameter to avoid suction effect. To calculate the percentage of the theoretical reference used values described by Morales [12].

A descriptive study was conducted of the entire group and separated by sexes, including mean, standard deviation (SD), range and percentiles level 25, 50 and 75%, using the Statistical Package for Social Science (SPSS 9.0 for Windows®) In comparisons of means he was used the Student t test, valuing equality of variances with the Levene test. For qualitative variables the Chi-square test was performed with Yates correction and Fisher's exact test. Correlations were established by Pearson r. The level of statistical significance was of $p<0.05$.

Results

We studied 171 patients with MO, of which 80 (47%) were men and 91 (53%) women. They have a history of smoking 27% of them with predominance of males (42% of men versus 13% of women). Our patients have an average weight of 130 Kg (SD=22), a BMI of 48 kg/m^2 (SD=6) and an average rate of wai/hip<1. The patient characteristics are expressed in Table 1.

More than half of patients have KCO, FRC, PIM and PEM below the limit of confidence of their theoretical values (Table 2), with no differences between the sexes, except for the maximum pressures. Men have less and less women PIM PEM (Table 3).

55% of patients, 57% of men and 54% of women ($p<0.08$), have hypoxemia in 10% being serious. Hypercapnia has 15% of cases: 18% of men and 13% of women ($p<0.0001$). The presence of hypercapnia and hypoxemia day in the OM is not related to weight or the type of obesity, but with older and decrease of forced ventilatory flows and static volumes (Table 4). However, this relationship is different depending on whether men or women (Table 5). There is a correlation between the degree and type of obesity and some parameters of lung function, which is small but statistically significant (Table 6).

	Men (n=80)	Women (n=91)	P value (p<)
Age (years)	42 ± 12	46 ± 12	0.03
Weight (kilos)	137 ± 22	123 ± 20	0.0001
Height (cm)	171 ± 30	158 ± 23	0.0001
BMI (Kg/m^2)	46 ± 6	48 ± 6	0.04
Waist (cm)	140 ± 15	136 ± 15	0.1
Hip (cm)	137 ± 14	142 ± 14	0.05
Waist/hip	1.025 ± 0.087	0.954 ± 0.094	0.0001
BMI: Bosy mass index			

Table 1: Anthropometric parameters in the MO population subject to study.

	Mean	SD	CV	Rank	Percentile 25	Percentile 50	Percentile 75
FVC (%)	97	15	15	66-144	87	98	106
FEV_1 (%)	93	17	18	57-152	83	95	102
FEV_1/FVC	80	7	9	53-95	77	81	85
D_LCO (%)	84	16	19	39-120	75	87	94
KCO (%)	76	13	17	46-100	67	77	88
TLC (%)	98	13	13	65-149	90	96	105
RV (%)	88	26	30	21-155	67	85	105
FRC (%)	71	18	25	38-120	58	69	81
MIP (%)	68	22	32	25-128	53	66	82
MEP (%)	62	19	31	34-122	49	61	74
PO_2 mmHg	78	15	19	41-116	69	77	85
PCO_2 mmHg	42	5	12	29-61	39	41	44

Sat. O_2 (%)	93	4	4	73-98	92	94	96

SD: Standard Desviation; CV: Coefficient of Variation; FVC: Forced Vital Capacity;

FEV1: First Second Forced Expiratory Volume; TLC: Total lung capacity; FRC: Functional respiratory capacity; RV: Residual volume; DLCO: Diffusing capacity for carbon monoxide; KCO: DLCO/ alveolar ventilation; MIP: Maximum inspiratory pressure; MEP: Maximum expiratory pressure.

Table 2: Blood gas and respiratory functional parameters in the MO.

	Men	**Women**	**P value (p<)**
FVC (%)	95 ± 12	100 ± 17	0.06
FEV1 (%)	91 ± 14	96 ± 19	0.1
FEV1/FVC	80 ± 7	81 ± 7	0.3
D_LCO (%)	86 ± 16	82 ± 15	0.2
KCO (%)	77 ± 13	76 ± 14	0.9
TLC (%)	98 ± 14	98 ± 13	0.9
RV (%)	88 ± 29	88 ± 22	0.9
FRC (%)	72 ± 20	71 ± 16	0.7
MIP (%)	59 ± 15	77 ± 24	0.0001
MEP (%)	68 ± 19	54 ± 14	0.001
PO_2 (mmHg)	78 ± 15	78 ± 14	0.7
PCO_2 (mmHg)	42 ± 5	42 ± 5	0.9
Sat. O_2 (%)	93 ± 4	93 ± 4	0.8

FVC: Forced Vital Capacity; FEV1: First Second Forced Expiratory Volume; DLCO: Diffusing Capacity for Carbon Monoxide; KCO: DLCO/Alveolar Ventilation; TLC: Total Lung Capacity; RV: Residual Volume; FRC: Functional Respiratory Capacity; MIP: Maximum Inspiratory Pressure; MEP: Maximum Expiratory Pressure;

Values expressed as mean ± standard desviation.

Table 3: Comparison of respiratory functional parameters among men and women with MO

	Age (years)	**FVC (%)**	**FEV1 (%)**	**FEV1/FVC**	**RV (%)**	**FRC (%)**
Hypercapnia	47 ± 13	92 ± 16	86 ± 18	78 ± 7	98 ± 27	76 ± 18
Normocapnia	43 ± 12	95 ± 16	95 ± 16	81 ± 7	85 ± 26	70 ± 18
P value (p<)	0.2	0.04	0.01	0.04	0.04	0.1
Severe hypoxemia	48 ± 11	94 ± 16	90 ± 17	79 ± 7	94 ± 24	74 ± 18
No Severe hypoxemia	39 ± 12	102 ± 12	99 ± 15	82 ± 6	78 ± 28	67 ± 18
P value (p<)	0.0001	0.003	0.002	0.02	0.005	0.05

FVC: Forced Vital Capacity; FEV1: First Second Forced Expiratory Volume; RV: Residual Volume;

FRC: Functional Respiratory Capacity; Values expressed as mean ± standard desviation.

Table 4: MO: comparisons between patients with severe hypoxemia and hypercapnia and normal.

	Hypercapnia	**Normocapnia**	**P value (p<)**	**Severe hypoxemia**	**No severe hypoxemia**	**P value (p<)**
Men						

Age (years)	43 ± 14	41 ± 12	0.7	46 ± 11	36 ± 12	0.001
FVC (%)	87 ± 14	98 ± 11	0.003	91 ±13	102 ± 9	0.001
FEV^1 (%)	82 ± 16	94 ± 12	0.01	87 ± 15	97 ±11	0.004
RV (%)	98 ± 31	85 ± 29	0.1	96 ± 27	77 ± 31	0.02
KCO (%)	82 ± 12	74 ± 13	0.05	77 ± 13	75 ± 13	0.4
Women						
Age (years)	53 ± 9	45 ± 12	0.03	50 ± 11	41 ± 11	0.001
FEV1/FVC	79 ± 5	81 ± 7	0.3	80 ± 7	83 ± 6	0.05
FRC (%)	84 ± 16	68 ± 15	0.03	74 ± 14	66 ± 17	0.8
FVC: Forced Vital Capacity; FEV1: First Second Forced Expiratory Volume; RV: Residual Volume; KCO: DLCO/ Alveolar Ventilation; FRC: Functional Respiratory Capacity; Values expressed as mean ± standard desviation.						

Table 5: Comparisons, depending on the presence of hypoxemia and hypercapnia, in men and women with MO.

		PCO_2	FVC	Tiff	TLC	MEP
BMI	r	0.143	-0107	0.169	-0.078	-0.244
(Kg/m^2)	p	0.08	0.2	0.05	0.4	0.03
Waist	r	0.195	-0.322	0.108	-0.350	-0.058
(cm)	p	0.07	0.005	0.3	0.006	0.6
Hip	r	0.229	-0.165	0.087	-0.341	-0.188
(cm)	p	0.03	0.1	0.4	0.007	0.1
waist/hip	r	-0.031	-0.215	0.050	-0.081	0.134
	p	0.7	0.06	0.6	0.5	0.3
BMI: Body Mass Index; FVC: Forced Vital Capacity; Tiff.: FEV1/FVC; TLC: Total Lung Capacity; MEP: Maximum Expiratory Pressure.						

Table 6: Correlation between parameters determining the type of obesity and blood gas and respiratory functional parameters in MO.

Discussion

It is accepted that obese subjects have a restrictive ventilatory pattern type with decreased lung volumes and ventilation/perfusion ratio with lower closing volume in the pulmonary regions declines, which condition an increase in the difference alveolar-arterial of O_2 [13].

In patients with OM it has also been reported that restrictive ventilatory pattern is attributed to increased abdominal volume, the displacement of the diaphragm, the greater weight of the chest wall and the deposit of fat in both the diaphragm and intercostal muscles. Thus, the MO causes a reduction in lung volumes, especially at the level of the ERV, which is reversible with ponderal loss [14]. This occurs in half of our patients who have less than 70% of its theoretical value FRC. ERV likely decrease as they maintain their values RV and TLC, but with great variability. The so-called dynamic volumes, obtained by spirometry, are in the range of normal for the entire group. Men with higher incidence of smoking, have lower FVC, which does not become significant.

Pulmonary mechanical alterations caused by obesity produce an increase in alveolar-arterial oxygen difference that determines a lower PO2 [15]. Thus, there has been a high prevalence of hypoxemia in obesity has been linked to a decreased volume of closing and alteration in the distribution of ventilation [16]. In our study, 75% of the cases had a PO2 lower to 85 mmHg and 25% PO_2 below to 70 mmHg, in adult patients without respiratory disease, with an average age of 44 years. The prevalence of hypoxemia is similar between men and women. Hypercapnia is observed more in men, perhaps due to the prevalence of obesity and lower central rate PEM, which could condition worse muscular efficiency, especially the diaphragm.

In obese patients it has not been stated clearly why some have hypercapnia and others not. Hypercapnia has been associated with decreased TLC, VC and FEV1, unrelated to age, BMI, or obesity [17]. Our patients with MO and hypercapnia have lower FVC, FEV1 and FEV1/FVC and higher RV, with no differences in muscle pressures. There may be in the genesis of hypercapnia mechanical alteration of the chest wall, with varying degrees of air trapping. There is no correlation between hypercapnia and BMI and waist circumference. The decrease in forced expiratory flows in men with hypercapnia does not occur in women.

Published values of DLCO in obesity are contradictory. While a decrease in DLCO study demonstrated in 20% of the obesity patients [18], another 10% increase in DLCO and 20-25% in the KCO [18] are observed. It has also described a significant increase in DLCO after a weight loss important [6]. In our patients with MO DLCO it remains within the normal range, although the KCO is decreased in half of the cases. DLCO and KCO are similar in both sexes, without having correlation with the extent or type of obesity.

It is believed that the distribution of body fat influences the ventilation in men and that this influence is modified with age. Described in males overweight (mean BMI 26 kg/m^2), under 60 years, obesity central rate, defined according to the index wai/hip and thickness subscapularis fold in a lower FVC and FEV1. Android type obesity causes a greater involvement of static volumes, demonstrated by the inverse relationship between volume and static measurement biceps skinfold thickness or wai/hip index, after excluding smoking as a confounder. The upper body fatness, measured as wai/hip or more

index of 0.950, is also related to a decrease in dynamic volumes [5]. Similarly, our morbidly obese patients with increased waist have lower FVC and TLC. However, no correlation with cin/cad index [19].

The reduction in lung volumes obesity causes increased resistance and lower chest wall respiratory distensibility [20] without increased maximal respiratory pressures. Thus, although both measures maxims as FRC in patients with MO, respiratory muscle pressures are usually lower than in non-obese subjects, for both men and women, these differences are not significatives [21]. Our results, referring to a Mediterranean population, show greatly diminished percentage values of PIM and PEM. This decrease behaves differently according to sex. Thus, men have higher PEM than women, probably by increased expiratory muscle strength and lower obesity but lower PIM that they, perhaps because diaphragmatic dysfunction, since the residual volume is similar in both groups. No correlation between respiratory pressures and type of obesity.

We conclude that in morbid obesity is a widespread decline in flows, volumes and respiratory maximum pressures, with frequent blood gas involvement occurs. There are differences between men and women for the presence of hypercapnia and respiratory maximum pressures. The type of obesity, measured as an wai/hip index, has little relevance in these alterations.

Acknowledgment

To Mr. Vicente Abella, Ms. Carmen Sempere and Ms. Pilar García for his estimable collaboration in the conduct of the respiratory function.

References

1. Spanish Society for the Study of Obesity (SEED) (2000) SEEDO'2000 consensus for the evaluation of overweight and obesity and establishment of criteria for therapeutic intervention. Med Clin (Barc) 115: 587-597.
2. Spanish Society for the Study of Obesity (SEED) (1996) Spanish consensus 1995 for evaluation of obesity and epidemiological studies. Med Clin (Barc) 107: 782-787.
3. Sugerman HJ (1987) Pulmonary function in morbid obesity. Gastroenterol Clin North Am16: 225-237.
4. Collins LC, Hoberty PD, JF Walker, Fletcher CD, Peiris AN (1995) The effect of body fat distribution on pulmonary function test. Chest 107: 1298-1302.
5. Lazarus R, Sparrow D, Weiss ST (1997) Effects of obesity and fat distribution on ventilatory function. The Normative Aging Study. Chest 111: 891-898.
6. CS Ray, Sue DY, Bray G, Hansen JE, Wasserman K (1983) Effects of obesity on respiratory function. Am Rev Respir Dis 128: 501-506.
7. Flenley DC (1980) Arterial puncture. Br Med J 12: 128-131.
8. Quajer Ph H, Tammeling GJ, Cotes JE, Pedersen OF, Perlin R, et al. (1993) Lung volumes and forced ventilatory flows. Report working party. Standardization of lung function test. European Community for steel and oral official statement of the European Respiratory Society. Eur Respir J 6: 5-40.
9. American Thoracic Society (1995) Single-breath carbon monoxide diffusing capacity (transfer factor): recommendation for a standard technique-1995 update. Am J Respir Crit Care Med 152: 2185-2198.
10. Quanjer PH (1983) Standardized testing lung function. Eur Respir Physiopathol Bull 5: 1-95.
11. Black LF, Hyatt RE (1969) Maximal respiratory pressure: Normal values and relationship to age and sex. Am Rev Respir Dis 99: 696-702.
12. Morales P, Sanchis J, PJ Lamb, Diez JL (1997) Maximum static pressure in adults. Reference values of a population Caucasian Mediterranean. Arch Bronconeumol 33: 213-219.
13. Chodoff P, Margand PMS, Imbembo AL (1974) Applied pulmonary physiology: morbid obesity and pulmonary function. Crit Care Med 2 : 123-128.
14. Thomas PS, Cowen ER, Hulands G, JS Milledge (1989) Respiratory function in the morbidly obese before and after weight loss. Thorax 44: 382-386.
15. Jenkins SC, Moxham J (1991) The effects of obesity on lung function mild. Respir Med 85: 309-311.
16. Rorvik S, Bo G (1976) Lung volumes and arterial blood gases in obesity. Scand J Respir Dis Suppl 95: 60-64.
17. Laaban JP, Orvoen-Frija E, D Cassuto, Pascal S, D Leger, et al. (1996) Mechanisms of diurnal hypercapnia in sleep apnea syndromes associated with morbid obesity. Presse Med 25: 12-16.
18. Prefaut C, Ramonatxo M, Monnier L, Mirouze J, Chardon G (1980) Mechanical effects of obesity on lung function. Pathol Biol (Paris) 28: 149-154.
19. Collard P, Wilputte JY, Aubert G, Rodenstein DO, Frans A (1996) The single-breath diffusing capacity for carbon monoxide in obstructive sleep apnea and obesity. Chest 110: 1189-1193.
20. Zerah F, Harf A, Perlemuter L, H Lorino, Lorino AM, Atlan G (1993) Effects of obesity on respiratory resistance. Chest 103: 1470-1476.
21. Kelly TM, Jensen RL, Elliott CG, Grapo RO (1988) Maximum Pressures in morbidly obese respiratory subjects. Respiration 54: 73-77.