

Computerised Spirometric Evaluation of Pulmonary Functions in Obese Hypertensive and Obese Non-Hypertensive Male Subjects; A Comparative Study

Anita Sreedharan^{1*}, Swarnalatha Pallikara Konath², Sanjeev Kumar Madakkavil³, Prasad Surendran⁴

¹Assistant Professor, Department of Physiology, Kannur Medical College, Anjarakandy Kerala, India

²Professor, HOD, Department of Physiology, Kannur Govt Medical College, Pariyaram Kannur Kerala, India

³Consultant Pulmonologist, Indira Gandhi Co operative Hospital Thalassery Kannur Kerala, India

⁴Cardio thoracic Vascular Surgeon HOD, Department of CardioThoracic Surgery, ASTER MIMS Hospital Kannur Kerala, India

Received: 05-07-2020 / Revised: 22-08-2020 / Accepted: 30-08-2020

Abstract

Background: Obesity has become a worldwide epidemic and is associated with impaired quality of life. It is now a major health concern in our country contributing significantly to increased morbidity and mortality, reducing life expectancy markedly among adults, and is commonly associated with hypertension. It causes mechanical obstruction of various parts of the body, including the respiratory system. **Materials and Methods:** A Total of three hundred (300) age and sex matched subjects were selected from those attending the Executive Health Check up OPD of kannur Medical College, which included One hundred (100) normal subjects (Group 1), One hundred (100) obese normotensive subjects (Group 2), and One hundred (100) obese hypertensive subjects (Group 3). **Results:** In the obese normotensive group and obese hypertensive group, statistically significant decline in FVC was seen as compared to the non-obese group. But there was no significant change in FVC between the hypertensive obese and normotensive obese groups. FEV1 showed no significant difference between regular, obese normotensive and obese hypertensive groups, although the obese groups showed a small insignificant decrease. FEV₁/FVC % did not show any significant change in the three groups. PEF did not show any significant change in the three groups. **Conclusion:** It can be concluded that obesity causes mechanical restriction to respiratory movements which adversely affect pulmonary functions. Hypertension had no adverse effects on lung functions.

Keywords: Obesity, Pulmonary Functions, Hypertension.

This is an Open Access article that uses a fund-ing model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided the original work is properly credited.

Introduction

Obesity is a public health problem worldwide, with a growing occurrence and prevalence and just over a century old. The impact of obesity on the quality of life was recorded in the 18th century, but it was recognised as a cause of disintegrating health only in the middle of the 19th century, and its adverse role in increased morbidity and mortality was documented only in the

first decades of the 20th century. The exponential rise in obesity incidence has led the World Health Organization to label it a Global Epidemic over the last sixty years. The indolent development of complications-hypertension, diabetes mellitus and atherosclerosis is responsible for morbidity and mortality[1]. Obesity is one of the most prevalent forms of malnutrition and is a major contributor to ill health. It is defined as an abnormal growth of adipose tissue due to an increase in fat cell size (hypertrophic obesity) or an increase in the number of fat cells (hyperplastic obesity), or a combination of the two. Obesity is frequently expressed in terms of the Body Mass Index (BMI), a simple index used to classify adult obesity, overweight, and underweight. BMI is defined as the weight in kilograms

*Correspondence

Dr. Anita Sreedharan

Assistant Professor, Department of Physiology, Kannur Medical College, Anjarakandy Kerala, India.

E-mail: aniprasad65@yahoo.in

divided by the square of the height of the person in metres (kg/m^2). BMI $> 30.0 \text{ kg}/\text{m}^2$ is classified as obesity and the BMI between 18.50 to 24.99 kg/m^2 is taken as the normal range[2]. Obesity gives additional strain to the cardio- respiratory system[3]. It is linked to hypertension, dyslipidemia, ischemic heart disease, diabetes mellitus, osteoarthritis, liver disease and respiratory anomalies[4]. As far as respiration is concerned, birth involves sudden transfer from a situation in which no breathing effort is necessary to one in which continued breathing effort is indispensable[5]. Obesity can alter respiratory physiology leading to resistance abnormalities, ventilation and perfusion relationships, respiratory muscle workload, upper airway calibre & tone, ventilation control, and Compliance of pulmonary and chest walls[6]. Obesity affects various respiratory parameters such as compliance, neuromuscular strength, breathing work, spirometric measurements, respiratory resistance, diffusing capacity and exchange of gases, and reduces respiratory muscle strength and respiratory muscle efficiency, particularly that of the diaphragm. A possible cause of impaired respiratory function in obesity includes increased elastic load which must be overcome by the respiratory muscles during inspiration. An overstretched diaphragm would place this respiratory muscle at a mechanical disadvantage, resulting in reduced muscle strength and efficiency of inspiration. Extremely obese subjects have a tendency towards rapid and shallow breathing to overcome the reduced total respiratory compliance and inefficiency of the respiratory muscles. Obesity increases the oxygen consumption by the muscles and the airway resistance. Obese people may be predisposed to respiratory failure during situations that tax the respiratory system further[7], As age advances there is variability in pulmonary functions such as Forced Vital Capacity (FVC), Forced Expiratory Volume in 1st Second (FEV₁), Functional Residual Capacity (FRC), Residual Volume (RV), etc[8]. The changes in the parameters of the pulmonary function are mild, but may become significant in people with obesity. The study conducted by Rubinstein I et al in obese, non-smoking men found a restrictive ventilatory impairment, with reduction in forced expiratory volume in 1st second (FEV₁), Forced Vital Capacity (FVC), Total Lung Capacity (TLC), Functional Residual Capacity (FRC), and Expiratory Reserve Volume (ERV)[9]. Obesity and weight gain are independent risk factors for developing hypertension. Hypertension is associated with weight gain in people with previously normal blood pressure and lowering of

arterial blood pressure on weight loss is seen in hypertensive obese persons[10]. Clinic blood pressure of 140/90 mm Hg is recommended for diagnosing hypertension[11]. Systemic hypertension leads to left ventricle pressure overload which predisposes to concentrated left ventricular hypertrophy occurring with volume overload conditions alone. Mixed pressure and volume overload leads to increased left ventricle wall stress. Diastolic dysfunction is common in obese subjects, and the risk of systolic dysfunction increases with coexisting hypertension. Systolic dysfunction or cardiomyopathy due to obesity results if the degree of hypertrophy does not keep pace with that of chamber dilatation[12]. Pulmonary function tests are an essential part of clinical practice[13]. Spirometry is the most common method for measuring pulmonary function tests. Lung functions especially the amount (volume) and/or speed (flow) of air that can be exhaled or inhaled can be measured[14]. Obesity is considered the most common factor that affects the relationship between the lungs, chest wall and diaphragm, leading to significant affects in pulmonary function values that can be measured by spirometry. Shashi Mahajan et al. studied the pulmonary function tests of normal non-obese Amritsar males and obese Amritsar males and found a decrease in obese group Forced Vital Capacity compared to non-obese group[15]. In the present study, an attempt will be made to study the adverse effects of obesity and hypertension on selected parameters of pulmonary function tests using a computerised spirometer.

Objectives

- To study the effects of obesity on pulmonary functions FVC, FEV₁, FEV₁/FVC% and PEF in 40-60 year old obese, non-hypertensive male subjects comparing them with normal controls using a computerised spirometer.
- To study the effects of obesity on pulmonary functions FVC, FEV₁, FEV₁/FVC% and PEF in 40-60 year old obese, hypertensive male subjects comparing them with controls using a computerised spirometer.
- To study the effects of hypertension on pulmonary functions FVC, FEV₁, FEV₁/FVC%, and PEF in 40-60 year old obese, hypertensive male subjects comparing them with obese normotensive male subjects of the same age group.

Materials and methods

This study is a comparative study of pulmonary functions of obese normotensive male subjects and obese hypertensive male subjects with normal male subjects of 40-60 years age group. Study was conducted in the Department of Physiology and Department of Chest Medicine, Kannur Medical College, Anjarakandy, Kannur, on subjects who came to Kannur Medical College Hospital OPD for Executive Health Check-up. Pulmonary functions tests were performed on 300 male subjects who were divided into 3 groups.

- 100 normal subjects(group 1)
- 100 obese normotensive subjects(group 2) and
- 100 obese hypertensive subjects (group 3).

The differences in the values of parameters FVC, FEV₁, FEV₁/FVC% and PEF between the groups were analyzed and discussed.

Inclusion criteria

- Male subjects 40-60years of age with BMI between 18.5-24.9 kg/m² who are normotensive (assumed blood pressure ≤ 120mm Hg systolic and ≤ 80mm Hg diastolic)[16]
- Male subjects 40-60years with BMI ≥ 30kg/m², who are normotensive. Male subject's 40-60years with BMI ≥ 30kg/m²and are hypertensive (blood pressure ≥ 140/90mm Hg).

Exclusion criteria

- Male subjects with evidence of cardiopulmonary diseases.
- Female subjects.
- Smokers.

Ethical clearance was obtained from the Kannur Medical College Ethical Clearance Committee for Human Research to conduct the study. Health status of the subjects were ascertained through history taking and clinical examination before recording the spirogram. Written informed consent was taken from each subject before conducting the pulmonary function tests. Weight (kg) was measured using a calibrated weighing machine in light clothing and bare feet using the same weighing machine for all subjects and height (cms) was measured using a centimetre measuring scale attached to the wall. Using the formula BMI = Weight in kg / Height² (ms) was calculated.

Method of recording of Blood Pressure

Blood Pressure was recorded in resting state by using the same sphygmomanometer for all the subjects. Subjects with blood pressure ≥140mmHg systolic and ≥90 mmHg diastolic were taken as hypertensives and

they had their blood pressure taken in two sessions. The subjects' systolic and diastolic blood pressure was measured in the right arm using the sphygmomanometer in the lying position after a rest of 15 minutes. BPL ARPEMIS handheld Personal Computer based spirometer was used to test the lung functions. BPL ARPEMIS is a serial contact spirometer based on Windows 95/98. This spirometer has a mouthpiece attached to the transducer assembly which is connected by a cable to the Computer. Software of BPL ARPEMIS Healthcare group, BPL Ltd was installed in the computer. This software allows the predicted values for age, sex and height, and it also gives a recorded value of all the parameters. To enhance patient performance, BPL ARPEMIS has a unique feature, a WATER TANK for patient motivation. Since spirometry is a voluntary test, it requires maximum effort and co-operation from the patient. Lay people are neither used to nor aware of spirometry. When you input the relevant data, the screen changes to the FVC test. Instead of predicted values being displayed a water tank with 3 level colours will be seen. All you have to ask the patient to do is to fill the water tank with blue water with his expires when the red button turns to green. The more he expires the more respective levels are overwritten with blue water.

The level colours:

Red = 0-60% of predicted FVC.

Yellow = 60-80% of predicted FVC.

Green = 80-100% of predicted FVC.

After proper guidance and practise with nose clips in place and lips tightly placed around the sterilised and reusable mouthpiece in the sitting posture, the subject was asked to inspire with maximum effort and then expire quickly and with maximum force into the mouth piece. Three trials were performed and the best of the three spirograms was selected and the best value of the following parameters FVC, FEV₁, FEV₁/FVC% and PEF in each spirogram was considered for evaluation and comparison. The values of the various parameters are entered into the Microsoft Excel spreadsheet. BPL ARPEMIS is factory calibrated. It is recommended that calibration be done once in 6 months or as per usage.

Statistical Analysis

Data was entered in Microsoft Excel spreadsheet 2010. Data was analysed using the statistical software SPSS-17th version. Microsoft Word 2010 version was used to generate graphs and tables. Using ANOVA, Kruskal Wallis H Test and Mann Whitney U Test the parameters FVC, FEV₁, FEV₁ / FVC percent, and PEF were analysed. FEV₁ was analysed using ANOVA as the

assumption of normality was satisfied. FVC, FEV₁/FVC and PEF were analysed using Kruskal Wallis H Test to find the significant difference between the three groups. Mann Whitney U test was used for pairwise comparisons. A p-value less than 0.05 has been found statistically significant. Graphs are those of Median except FEV₁, which is that of Mean.

Results

The present study was conducted at the Department of Physiology and Chest Medicine, Kannur Medical College to record the pulmonary functions FVC, FEV₁, FEV₁ / FVC percent and PEF among 300 male subjects in the 40-60 year age group who attended Kannur Medical College OPD. The subjects of the study were split up into three classes. Groups 1, Groups 2 and 3. Group 1 consisted of 100 normal subjects with the following characteristics in Mean \pm SD.

Table 1: General characteristic of Group1 (normal) study subjects

Characteristics	Mean \pm SD
Age in years	50.14 \pm 6.65
Weight in Kg	62.84 \pm 8.63
Height in cms	163.98 \pm 6.78
BMI in Kg/m ²	23.33 \pm 2.54
SBP in mm Hg	123.14 \pm 9.32
DBP in mm Hg	78.50 \pm 5.91

Group 2 consisted of 100 obese normotensive subjects with the following characteristics

Table 2: General characteristics of Group 2 (obesity without hypertension) study subjects

Characteristics	Mean \pm SD
Age in years	49.64 \pm 6.73
Weight in kg	84.22 \pm 7.34
Height in cms	164.34 \pm 8.51
BMI in kg/m ²	30.98 \pm 1.65
SBP in mm Hg	127.70 \pm 7.97
DBP in mm Hg	81.66 \pm 5.01

Group 3 consisted of 100 obese hypertensive subjects with the following characteristics.

Table 3: General characteristics of Group 3 (Obesity with hypertension) study subjects

Characteristics	Mean \pm SD
Age in years	48.47 \pm 5.60
Weight in Kg	84.62 \pm 7.64
Height in cms	163.61 \pm 17.18
BMI in Kg/m ²	31.00 \pm 2.10
SBP in mm Hg	147 \pm 5.98
DBP in mmHg	93.20 \pm 3.27

Age was not statistically significant among the three groups. The three groups are similar in age (40 – 60 years) in terms of basic characteristics. Group 2 and 3 showed significant differences in BMI compared to Group 1. The basic characteristics in Tables 1, 2 and 3

indicate that there was statistically significant difference between group 1 and 3 subjects in SBP and DBP ($p < 0.001$) but no substantial difference between group 1 and group 2.

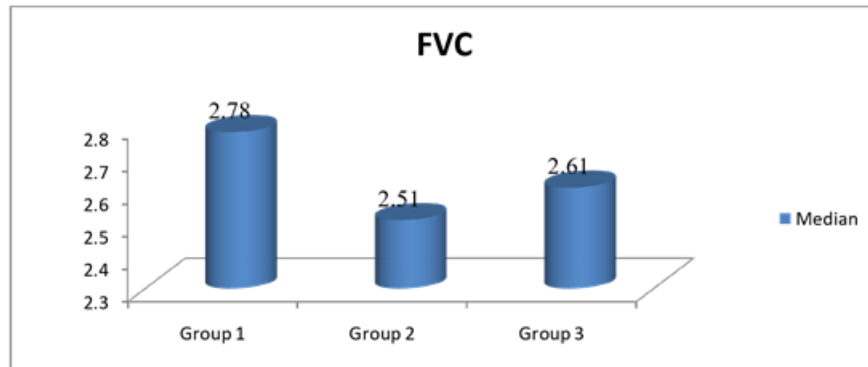


Fig 1: FVC

The FVC value of the three groups shown in Figure 1 was found to be significantly different. FVC was found to be higher in Group 1 relative to Group 2 and Group 3. The difference between Group 2 and Group 3 is not significant. Group 1 FVC values are significantly different from Group 2 (p -value < 0.001) and Group 3

(p -value 0.005). However, there is no substantial average difference in FVC between Group 2 and Group 3 should use as the p -value obtained for measuring variance between both groups is 0.068, which is > 0.05 . The range of median is expressed in terms of Inter Quartile Range (IQR)

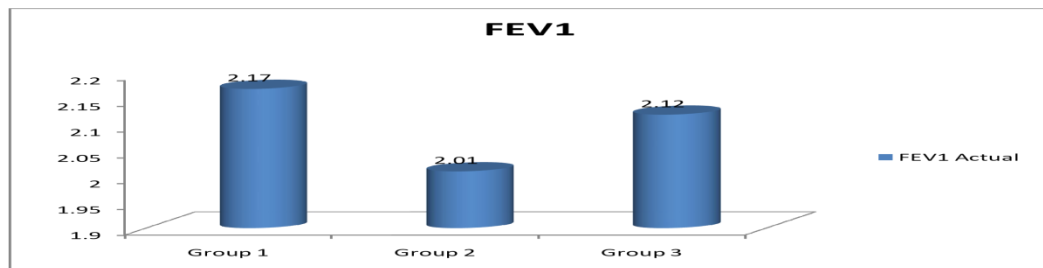


Fig 2: FEV₁

The FEV1 values between the 3 groups were not found to be significantly different. Between the regular group (group 1) and the obese groups (groups 2 and 3) there was a small insignificant decrease in FVC.

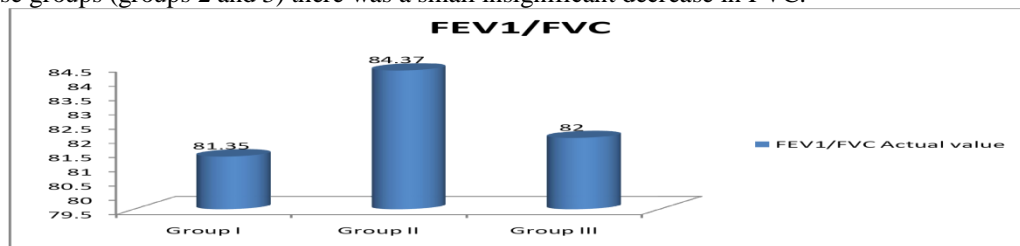


Fig 3: FEV₁/FVC

There was no substantial improvement in the percentage of FEV₁ / FVC between the three groups, and no

significant difference between the hypertensive obese and non hypertensive obese groups.

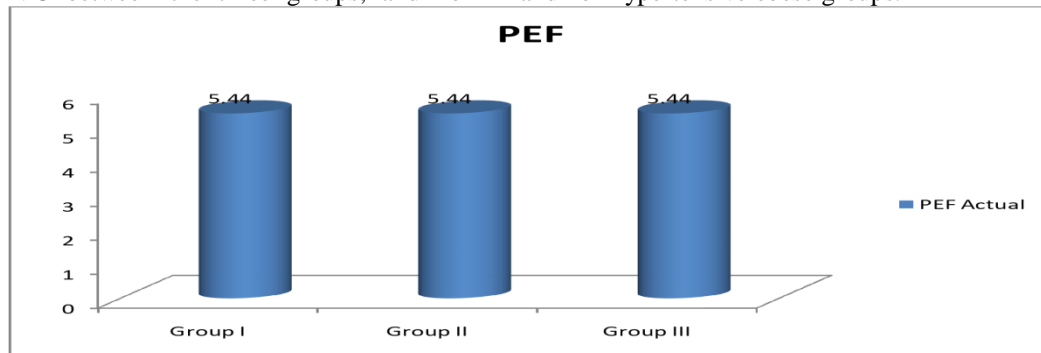


Fig 4: PEF

The PEF value was not significantly different in the three groups.

Discussion

In this study males of age group of 40-60 years were selected because of the influence of the middle-east, which is very vividly seen in the population of North Kerala. Males employed in the countries of the Middle East are often used for the unhealthy and irresponsible consumption of calorie-rich and easily available fast food and standardised working profile with a sedentary lifestyle, making them more likely to become obese. In the present research, a significant decrease in the level of Forced Vital Capacity (FVC) was noted in the obese normotensive and obese hypertensive groups 2 and 3 compared with their matched controls, group 1, and this confirms earlier studies. But the FVC did not indicate any significant difference between group 2 and group 3. The significant reduction in the level of FVC in the obese groups 2 and 3 compared to group 1 can be attributed to the restrictive effect of obesity. There was no significant reduction in lung function parameter FVC specifically due to hypertension possibly due to the fact that these are newly detected cases and long standing hypertension has effect on lung functions.

FVC and FEV₁

In support of this study, Ray CS et al[17], Refsum HE et al[18] and Bottai M et al[19] demonstrated an increase in lung function FVC by losing weight that indicates the negative effects of obesity, and that these effects do not involve permanent structural remodelling of the airways. Changes of reduced FVC similar to the present study was found in the study conducted by Rubinstein I et al. on obese non smoking men. This is thought to be a consequence of the mechanical strain on

the chest wall and diaphragm, which in turn decreases thoracic compliance and impedes diaphragmic descent. Rubinstein and colleagues have found that obese people have significantly higher airway resistance and lower FEV₁ compared to non-obese people. Restrictive impairment can be seen here [9]. Some studies by Carey IM et al. have also shown a similar inverse association between FVC and BMI, which is more likely to be due to a greater central distribution of fat in men than in women[20]. Earlier research by Biring MS et al. supports the adverse effects of obesity, in which significant improvements were found in the study on pulmonary physiological changes in obesity. FVC was reduced in the obese community and indicated that obesity could cause airflow restriction with reduction in both FVC and FEV₁ and that FEV₁ and FVC were symmetrically reduced in this study[21]. Study conducted by Raida I et al. on the socioeconomic factors or racial difference in lung function in Maryland showed that the reduced FVC observed in obesity was more in men than among women. Education had less impact than the index of poverty in accounting for the racial disparity between white and Afro-American women and BMI was inversely associated with FEV₁ and FVC[22]. Our studies showed only an insignificant reduction in FEV₁ like the study done in UK by Canopy D et al[23]. On the contrary, a research conducted in Saudi Arabia by Mohammed Al Hoban did not show reduced FVC and FEV₁, which may be due to wide variance in PFT values in the ethnicity of different populations or may be the result of differing methodologies in the research[24]. Our analysis shows a substantial reduction in FVC in the obese normotensive and obese

hypertensive groups compared with the usual group due to the impact of obesity but no significant difference in FVC between the two groups as hypertension does not appear to have any adverse effect here. Our study shows a slight reduction in FEV₁ in obese normotensive and obese hypertensive groups 2 and 3 compared to normal group 1 and, at the same time, there is no important difference between group 2 and 3 because the effect of hypertension on lung function is not observed. Low values of FVC and slightly reduced FEV₁ indicate a restrictive lung pattern among obese people. The cause of decline in these respiratory parameters in obesity may be due to reduced distensibility of the chest wall or limited expansion of the thoracic cavity.

FEV₁/FVC%

In a study performed on 103 obese men, Rubinstein I et al. found that the FEV₁ / FVC ratio is usually normal or increased[9]. This study was normal, as was the study described above. Possible cause can also involve obesity-related inflammation or small airway oedema, thus reducing their calibre. The increased ratio may be attributable to the peripheral closing of the airways and the resultant gas trapping, which may significantly reduce the FVC. The assumption is that, unlike in the present research, obesity can affect small airway function. A recent research by Leone et al. indicates that abdominal obesity may be associated with a reduced FVC / FEV₁ ratio suggesting an impact on the broad airway calibre, as well as suggesting that obesity may be associated with obstructive ventilatory abnormality in addition to its well-known association with restrictive abnormality[25]. Study by Biring MS et al[21] demonstrated a preserved FEV₁/FVC ratio which remains unaltered like this present study.

FVC and FEV₁ were symmetrically reduced in the obese group probably due to airflow limitation suggesting a restrictive pattern. Our study shows near normal values in the obese groups, group 2 and 3 on the average. Several longitudinal studies indicate that this ratio with obesity is normal, or increased. This is due to peripheral gas trapping due to the peripheral closure of the airways and thus a substantial reduction of the FVC. The assumption is that while obesity can affect small airway functions it does not affect big airways. Similar to this study, a recent analysis performed by Sin DD et al[26], FVC and FEV₁ are reduced symmetrically, resulting in a retained FEV₁ / FVC ratio consistent with a restrictive model. Here it has been indicated that obesity can cause airflow limitation with both FEV₁ and FVC reduction. Shashi Mahajan et al. study showed

negligible changes in FEV₁ / FVC, as in this present analysis. Low FVC and FEV₁ values indicate a restrictive pattern among obese people[15]. The cause of the decline in the respiratory parameters may be due to a reduction in the distensibility of the chest wall, causing changes in the volume and flow of ventilation. The study conducted by Mohammed Al Ghobain among the non-smoking obese and non-obese groups of the Saudi population did not show any significant differences in the FEV₁:FVC ratio between these two groups[24]. But in this study there was no significant reduction in FVC and FEV₁.

Peak Expiratory Flow (PEF)

This analysis shows no major difference in the PEF values. On the contrary, GG King et al's research showed a substantial reduction of the obese population, indicating the existence of air flow limitation in obese males[27]. Similar to this study, the study conducted by Shashi Mahajan et al[15] on lung functions in Amritsar's adult male population showed negligible changes in the obese group PEF values compared to normal groups. An association of hypertension with pulmonary functions can be expected because acute and chronic elevations of left atrial pressure due to hypertensive reduction of left ventricular compliance are known to cause elevation of pulmonary artery pressure which in turn may lead to a reduction in FEV₁ and FVC. The pulmonary functions FVC, FEV₁, FEV₁/ FVC percent and PEF are not reduced significantly specifically due to hypertension in this present study. Increased systemic and pulmonary vascular resistance and increased vessel stiffness can be associated with hypertension, but there is no substantial reduction in pulmonary functions FVC, FEV₁, FEV₁/FVC percent and PEF, possibly because these subjects are not long-standing hypertensive. Reduced FVC due to obesity precedes the onset of hypertension. Left ventricular thickness and therefore altered left ventricular preload to the left ventricular mass cause alterations in pulmonary function, however this only happens after long-standing hypertension. In this study there was a decline in FVC in obese normotensive and obese hypertensive subjects compared with standard controls. This observation indicates a restrictive pattern of obesity-related lung functions. There was no statistically significant difference between obese hypertensive and obese normotensive groups which means that hypertension does not play a significant role in this study. In this study, FEV₁ was mildly reduced in the obese groups suggesting a restrictive disorder and there was no significant effect of hypertension on the

pulmonary functions. Therefore significantly reduced FVC, insignificant difference in FEV₁, FEV₁/FVC ratio and PEF indicates that obesity results in restrictive pulmonary disease and there is less element of obstructive pulmonary diseases.

Conclusion

The following conclusion can be drawn from the results of the present study.

The FVC values in the Obese Normotensive and Obese Hypertensive Groups were substantially decreased relative to the regular group but there was no substantial difference between the Obese Hypertensive Group and the Obese Normotensive Group. There was no significant change in FEV₁ / FVC% Compared with controls, there was no significant change in PEF values in both categories. Therefore significantly reduced FVC and mild reduction in FEV₁ and insignificant changes in FEV₁/FVC and PEF implies that obesity could result in a restrictive pattern of pulmonary disease. This study provides a glimpse into the variety of alterations in pulmonary functions even in the absence of overt disease in obese people and shows the vulnerability of obese people to respiratory impairment. At the same time hypertension did not show any significant alteration in pulmonary functions. A multidisciplinary effort must be taken to tackle the obesity epidemic. One should choose the most health friendly foods among our traditions and customs. Several methods like yoga, meditation, Indian dance forms, martial arts, jogging etc will make physical activity fun. The hazards of fast food culture and awareness of the quality of food consumed should be instilled at the community level. The Hippocrates quote, "Everything in excess is opposed by nature" aptly describes how in pursuing the 'good life' we have created an environment and society that unintentionally promotes weight gain and obesity. We recommend an improvement in the lifestyle of the present day population which is slowly and surely slipping into the epidemic of obesity. Preventing obesity would definitely help to boost the quality of life and thereby strengthen our nation's economy.

References

1. Eknayan G. A history of obesity, or how what was good became ugly and then bad. *Adv Chronic Kidney Dis.* 2006;13(4):421-427.
2. Park K. Textbook of Preventive and Social Medicine. 19th Edition. Jabalpur: M/s Banarasidas Publishers; 2007. pp.332-34.
3. Anthony Seaton, Douglas Seaton, A Gordon Twitch. *Krofton and Douglas Respiratory Diseases.* (Vol 2). 5th Edition. Wiley India (p) Limited; 2003. p.674.
4. Sharmeen Lotia, Mark C Bellamy. Anaesthesia and Morbid Obesity- Continuing Medical Education in Anaesthesia. *Crit Care Pain.* 2008;8(5):151-156
5. Bijlani RL. *Understanding Medical Physiology.* 3rd Edition. New Delhi: Jaypee Brothers; 2004. 294.
6. Parameswaran K, Todd DC, Soth M. Altered respiratory physiology in obesity. *Can Respir J.* 2006;13(4):203-210.
7. Akshay Sood. Altered Resting and Exercise Respiratory Physiology in Obesity. *Clinics in Chest Medicine.* 2009;30(3):445.
8. Chan ED, Welsh CH. Geriatric respiratory medicine. *Chest* 1998;114:1704-33.
9. Rubinstein I, Zamel N, DuBarry L, Hoffstein V. Airflow limitation in morbidly obese, nonsmoking men. *Ann Intern Med.* 1990;112(11):828-832.
10. Kasper, Braunwald, Fauci, Hauser, Longo, Jameson. *Harrison's Principles of Internal Medicine.* (Vol 2) 16th edition. USA: McGraw Hill Medical Publishing Division; 2005. 1466.
11. Kasper, Braunwald, Fauci, Hauser, Longo, Jameson. *Harrison's Principles of Internal Medicine.* (Vol 2) 16th edition. USA: McGraw Hill Medical Publishing Division; 2005. 1468.
12. Alpert MA. Obesity cardiomyopathy: pathophysiology and evolution of the clinical syndrome. *Am J Med Sci.* 2001;321(4):225-236.
13. Warren M Gold. Pulmonary Function Testing. In: John F Murray, Jay A Nadel, Robert J Mason, V Courtney Broadder, eds. *Textbook of Respiratory Medicine.* (Vol 1). 4th Edition. Philadelphia (US): Elsevier Saunders; 2008. 671.
14. Kiraly A. History of Spirometry. *JPAHS.* 2005;4(1):1-7
15. Mahajan S., Kaur Arora AK, Gupta P. Obesity and Spirometric Status correlation in Adult male population of Amritsar. *National Journal of Physiology Pharmacology.* 2012; 2(2): 93-98.
16. Bonow, Mann, Zipes, Libby. *Braunwald's heart disease.* 9th edition. Gurgaon: Elsevier saunders; 2012. p.995.
17. Ray CS, Sue DY, Bray G, Hansen JE, Wasserman K. Effects of obesity on respiratory function. *Am Rev Respir Dis.* 1983;128(3):501-506.

18. Refsum HE, Holter PH, Løvig T, Haffner JF, Stadaas JO. Pulmonary function and energy expenditure after marked weight loss in obese women. *Int J obes* 1990;14(2):175-83.
19. Bottai M, Pistelli F, Di Pede F, et al. Longitudinal changes of body mass index, spirometry and diffusion in a general population. *Eur Respir J*. 2002;20(3):665-673.
20. Carey IM, Cork DG, Shartan DP. The effects of adiposity and weight gain on FEV decline in a longitudinal study in adults. *Int J Obes Relat Metab Disord*. 1999;23 (9): 979-85.
21. Biring MS, Lewis MI, Liu JT, et al. Pulmonary physiological changes of morbid obesity. *Am J Med Sci* 1999;318:293-7
22. Raida I, Harik Khan, Jerome L, Flag, Denis C Muller and Robert A Wise. The effect of Anthropometric and Socioeconomic factors or the Racial Difference in Lung Function. *Am J of Respiratory and Critical Care Medicine*. 2001;164 (9);1647-54.
23. Canoy D, Luben R, Welch A, et al. Abdominal obesity and respiratory function in men and women in the EPIC-Norfolk Study, United Kingdom. *Am J Epidemiol*. 2004;159(12):1140-1149.
24. Al Ghobain M. The effect of obesity on spirometry tests among healthy non-smoking adults. *BMC Pulm Med*. 2012;12:10.
25. Leone N, Courbon D, Thomas F, et al. Lung function impairment and metabolic syndrome: the critical role of abdominal obesity. *Am J Respir Crit Care Med*. 2009;179(6):509-516.
26. Sin DD, Jones RL, Man SF. Obesity is a risk factor for dyspnea but not for airflow obstruction. *Arch Intern Med*. 2002;162(13):1477-1481.
27. King GG, Brown NJ, Dibo C, Thorpe CW, Murray P et al. The effects of body weight on airway calibre. *Eur. Respir J*. 2005;25:896-901.

Source of Support: Nil

Conflict of Interest: Nil