ORIGINAL RESEARCH

Comparison of PEFR and FEF (Lung Function Tests) between obese males and non-obese males

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ABSTRACT
Obesity has emerged as an important risk factor for chronic respiratory diseases such as chronic obstructive pulmonary disease (COPD), asthma, obstructive sleep apnea and obesity hypoventilation syndrome. It has emerged as an important risk factor for these respiratory diseases, and in many instances weight loss is associated with important symptomatic improvement. All subjects were explained about the procedures to be undertaken and written informed consent was obtained from them. A brief history, general physical examination and clinical examination of all the systems were done to exclude medical problems and to prevent confounding of results. The mean PEFR in obese male subjects was 5.89±0.85 (75.00±6.53% of percentage predicted). There was statistically significant decrease in the value of PEFR in obese male subjects compared to non-obese male controls ($p< 0.001$).

Keywords:PEFR, FEF, obesity

INTRODUCTION

The pulmonary function tests (PFTs) are the battery of tests that are used to assess the physiological respiratory efficiency of an individual. The factors that usually affect the values of pulmonary function tests are age, gender, height, weight, race or ethnicity, and possibly obesity.

Obese subjects with increased abdominal circumference have a greater risk of pulmonary impairment as seen in restrictive respiratory disorders [1].

In our study an attempt has been made to examine the effects of obesity on ventilator function. This may be useful for the success of the treatment in obese patients and to motivate them for weight loss and regular physical activity [2].

Studies showed FVC and FEV1 in the females correlated negatively to BMI and were significant but in males showed a negative correlation which is not statistically significant, hence present study is undertaken to investigate the pulmonary functions in obese males [3].

We aimed to evaluate the effects of obesity on PFTs among healthy young men by spirometry, because it is considered to be the initial screening tool for pulmonary diseases, most widely used, economic, and easy to conduct using equipment that is available in our pulmonology laboratory [4].

Obesity has emerged as an important risk factor for chronic respiratory diseases such as chronic obstructive pulmonary disease (COPD), asthma, obstructive sleep apnea and obesity hypoventilation syndrome. It has emerged as an important risk factor for these respiratory diseases, and in many instances weight loss is associated with important symptomatic improvement. Moreover, obesity may influence the development and presentation of these diseases [5].

In any given individual the effects of obesity on respiratory function depend on the mass and anatomical distribution of the excessive adipose tissue on the thorax and abdomen. The nature of the physiological impairment induced by obesity will vary with its extent and will ultimately reflect integrated abnormalities of ventilator mechanics/muscle function, pulmonary gas exchange, ventilatory control and cardiac performance. These respiratory derangements are consistently accentuated on adopting the supine posture [6].

METHODOLOGY

60 obese and 60 non-obese males in the age group of 25-40 years were selected randomly from the general population for Spirometric evaluation after obtaining informed consent.

INCLUSION CRITERIA

- Obese males aged 25-40 years.
- Non-obese males aged 25-40 years.

EXCLUSION CRITERIA

- Age below 25 years and above 40 years
- Subjects with a history of Asthma, Diabetes mellitus, hypertension, other cardiovascular diseases, chest disease or deformity, endocrine diseases or surgery.
- Subjects on chronic medication.
- Smokers
- Alcoholics
- Subjects with noticeable weight gain or weight loss over the preceding 3 months.

Based on this information by using tables of sample size, the sample size was found to be 60 subjects in each study and control group.

The criteria for obesity were taken on the basis of body mass index as per the standard protocol.

Height (m) and weight (kg) of the subjects will be recorded and BMI calculated as

$$\text{Body mass index} = \frac{\text{Weight (Kilogram)}}{\text{Height}^2 \text{ (meter)}} \quad \text{[Quetelet's index]}$$

Subjects will be classified into 2 groups based on BMI as follows:

Normal- BMI 18.5-25 kg / m2

Obese- BMI >30 kg / m2.

A structured Performa was used to collect the relevant information.

All subjects were explained about the procedures to be undertaken and written informed consent was obtained from them.

A brief history, general physical examination and clinical examination of all the systems were done to exclude medical problems and to prevent confounding of results.

The following tests will be performed in a sitting position 2-3 hours after light breakfast in sequence after familiarizing the subjects with the testing procedures.

LUNG FUNCTION TESTS

The following lung parameters will be measured:

- Peak expiratory flow rate (PEFR) in litres / second
- Mean forced expiratory flow during the middle half of the FVC ($FEF_{25-75\%}$) in litres/second.

The results for each parameter were compared between the obese and non-obese men and statistically analyzed.

STATISTICAL ANALYSIS: Correlation between BMI and all other parameters will be assessed by calculating Pearson's correlation co-efficient. The data will be compared using the student's Un-paired t test at 95% confidence level.

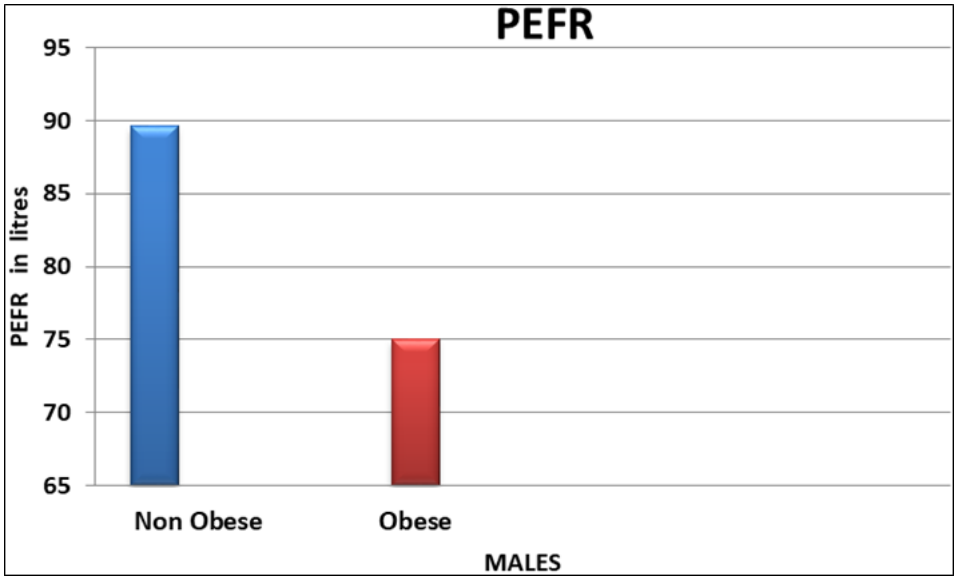
RESULTS

Table 1: Comparison of PEFR between Non Obese and Obese Males

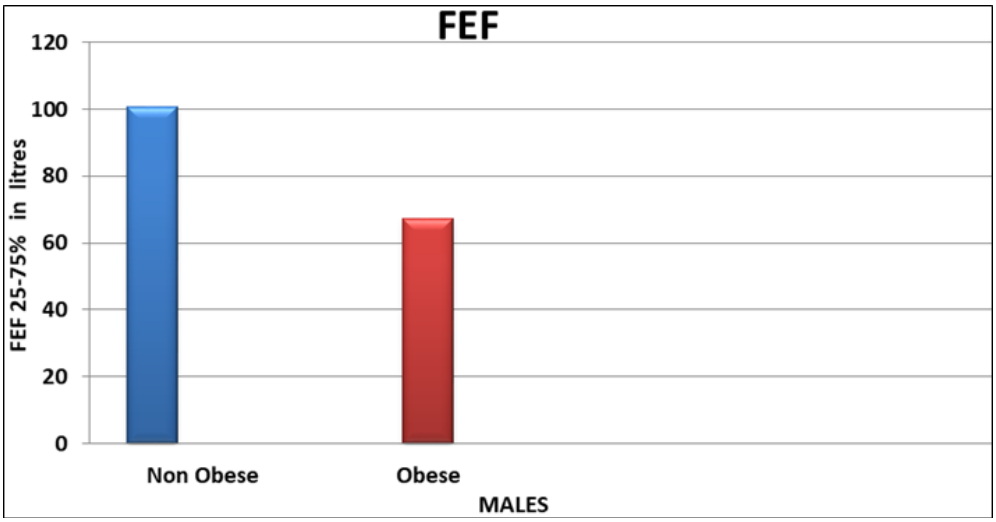
Groups	n	Actual value(L)		% Predicted	
		Range	Mean ± SD	Range	Mean ± SD
Non -Obese	60	7.12-10.28	8.40±0.73	47-110	89.63±9.99
Obese	60	4.28-9.38	5.89±0.85	62-102	75.00±6.53
Mean Difference		2.51		14.63	
Significance	t-value	17.42		9.49	
	p-value	0.000		0.000	

Table 2: Comparison of FEF 25-75% between Non Obese and Obese Males

Groups	n	Actual value(L)		% Predicted	
		Range	Mean ± SD	Range	Mean ± SD
Non -Obese	60	2.73-7.69	4.65 ± 1.05	62-152	100.77±20.69
Obese	60	2.03-6.01	2.57± 0.67	53-122	67.43 ± 10.71
Mean Difference		2.08		33.33	
Significance	t-value	12.902		11.10	
	p-value	0.000		0.000	



Graph 1: PEFR IN Obese subjects and Non obese controls



Graph 2: FEF IN Obese subjects and Non obese controls

PEFR (L/sec)

The mean PEFR in non-obese male controls was 8.40±0.73 (89.63±9.99% of the percentage predicted). The mean PEFR in obese male subjects was 5.89±0.85 (75.00±6.53% of percentage predicted). There was statistically significant decrease in the value of PEFR in obese male subjects compared to non-obese male controls ($p< 0.001$).

FEF 25-75% (L/sec)

The mean FEF25-75% in non-obese male controls was 4.65±1.05 (100.77±20.69% of the percentage predicted). The mean FEF25-75% in obese male subjects was 2.57±0.67 (67.43±10.71% of percentage predicted). There was statistically significant decrease in the value of FEF25-75% in obese male subjects compared to non-obese male controls ($p< 0.001$).

DISCUSSION

Physiological respiration is divided into two steps, process inspiration & process expiration. The two main muscles of inspiration are Diaphragm & External intercostals muscles. Two muscles of expiration are internal intercostals muscles & anterior abdominal wall muscles. The diaphragm belongs to the category of skeletal muscle fibers. Diaphragm consists of two domes & a central part called the tendinous portion of the diaphragm. This is the shape in resting condition.

During inspiration (I), stimulus from neurons of respiratory center located in medulla reaches the diaphragm through the spinal cord & phrenic nerve. Diaphragm now contracts, the domes are flattened & central tendon portion descends from thorax into the abdominal cavity. In resting condition the descent is 1-2 cm, in forced inspiration the diaphragm descends upto 8-10 cm. this increases ventral diameter of the chest wall.

Contraction of external intercostal muscles leads to bucket handle movement of the ribs which increases the transverse diameter of the chest wall. The above two movements result in pump handles movement of

sternum which increases thickness of the chest wall namely anterio - posterior diameter. The pressure inside the lungs called intra pulmonary pressure falls down severely resulting in process inspiration.

The expiration commences passively using elastic recoil property of the lungs. Expiratory muscles do not contract & (E) neurons of expiratory centre will not fire motor current in resting respiration but during deep breathing in inspiratory centre the (I) neurons fire more number of impulses resulting in stronger contraction of the diaphragm, which descends upto 8-10 cm from thoracic region into the abdominal cavity [7].

Now (E) neurons of expiratory center start firing motor current which reaches the muscles resulting in contraction of main expiratory muscles (Internal intercostals muscles & anterior abdominal wall muscles).

The most consistently reported effect of obesity on lung function is a reduction in the functional residual capacity (FRC). This effect reflects a shift in the balance of inflationary and deflationary pressures on the lung due to the mass load of adipose tissue around the rib cage and abdomen and in the visceral cavity. The reasons for the reduction are probably due to a mechanical effect of the adipose tissue. A reduction in the downward movement of the diaphragm, due to increased abdominal mass, is likely to decrease TLC by limiting the room for lung expansion on inflation [8].

Alternatively, deposition of fat in sub pleural spaces might directly reduce lung volume by reducing the volume of the chest cavity, although there is no direct evidence of any association between subpleural fat and either body fat or lung volumes. Evidence that respiratory muscle strength and maximum inspiratory and expiratory pressures are similar in obese and normal weight subjects suggests that stiffening of the chest wall is probably not a major determinant of TLC [9].

Abdominal and thoracic fat are likely to have direct effects on the downward movement of the diaphragm and on chest wall properties, while fat on the hips and thighs would be unlikely to have any direct mechanical effect on the lungs.

Obesity is characterized by a stiffening of the total respiratory system, which is presumed to be due to a combination of effects on lung and chest wall compliance. Reductions in lung compliance may be the result of increased pulmonary blood volume, closure of dependent airways, resulting in small areas of atelectasis, or increased alveolar surface tension due to the reduction in FRC [10].

Respiratory muscle function might also be impaired in obesity due to the mechanical disadvantage induced by changes in chest wall configuration, fat deposition and increased energy expenditure to expand the lungs, and an increase in intraabdominal adipose tissue which interferes with the mechanical properties of the chest wall causing decrease in compliance and preventing full excursion of the diaphragm [11].

Bronchoconstriction in the obese is associated with increased airway closure compared with nonobese controls and thus could increase gas trapping and alter ventilation distribution,

There are also effects of obesity on upper airway tone and hence resistance, which and a mechanical load that increases the work of breathing. Morbid obesity may also induce restrictive disturbance of respiratory function, related to reduced compliance of chest wall and or pulmonary parenchyma [12].

CONCLUSION

- The mean PEFR in obese male subjects was 5.89±0.85 (75.00±6.53% of percentage predicted). There was statistically significant decrease in the value of PEFR in obese male subjects compared to non-obese male controls.
- The mean FEF25-75% in obese male subjects was 2.57±0.67 (67.43±10.71% of percentage predicted). There was statistically significant decrease in the value of FEF25-75% in obese male subjects compared to non-obese male controls.

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