IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 15, Issue 3 Ver. VII (Mar. 2016), PP 01-05
www.iosrjournals.org

Effect of Weight Reduction on Peak Expiratory Flow Rate in Young Obese Individuals.

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I. Introduction

Obesity is a global phenomenon that increases morbidity and reduces life expectancy. There has been an increase in the prevalence of obesity [(1)]. With current trend of increasing obesity, it is expected that over 1 billion individuals will be obese by 2030 worldwide [(2)]. In India the prevalence of obesity ranges from 10-50%, in the 21stcentury, morbid obesity affecting 5% of the country's population. The overall prevalence of obesity was 6.8% (7.8 vs. 6.2%) and overweight 33.5% (35.0 vs. 32.0%) among women and men, respectively [(3)]. The highest prevalence of obesity (7.8%) and overweight (36.9%) was found among subjects aged 35-44 years in both sexes [(3)]. It has now become an important health problem in developing countries particularly in India [(4)]. Obesity is a chronic disease characterized by excessive body fat that causes damage to the individual's health [(5, 6)] and is associated with co morbidities such as diabetes [(7)], hypertension [(7, 8)], vascular dysfunction [(9,10)] and cardiac disease. Obesity, through its effect on chest and diaphragm is expected to modify respiratory functions [(11)].But it is not studied extensively and needs focused attention.

Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is defined as a person's weight in kilograms divided by the square of his height in meters (kg/m^2).The WHO definition is a BMI greater than or equal to 25 is overweight, a BMI greater than or equal to 30 is obesity. BMI provides the most useful population-level measure of overweight and obesity as it is the same for both sexes and for all ages of adults. The normal BMI range is between 18.5 to 24.99kg/m^2 [(5,6,7)].

Obesity has a profound effect on the physiology of breathing [(7)].Obesity can lead to pulmonary compromises in a number of ways, including decreases in respiratory compliance due to mechanical factors such as increased weight on the thoracic cage and abdomen, as well as changes in lung compliance. Massive weight loss after the bariatric surgery in obese patients proved to be beneficial for the lung function in many aspects [(12)], but in a long term pulmonary functions may have detrimental effects [(13)].Also The National Institute of Health has set patient criteria for weight loss surgery which states that eligible patients should have a BMI of 40 or higher (morbidly obese) or a BMI of at least 35 with co-morbidities (obesity related health conditions) such as diabetes, sleep apnea, heart disease, or hypertension. Before suffering from co-morbidities if proper reduction of weight is done, one can prevent obesity related health problems. Considering all these facts, this topic was chosen to study the effects of gradual weight reduction on pulmonary functions, particularly peak expiratory flow rate in obese individuals.

Aim and objectives

Aim: - To study effect of gradual weight reduction in groups with different weight loss targets on peak expiratory flow rate.

Objectives:-

1) To study peak expiratory flow rate before target weight reduction in obese individuals.
2) To study peak expiratory flow rate after target weight reduction in obese individuals.
3) To compare them and find out if any significance is observed in different target reduction groups.

II. Pathophysiology

Body weight is regulated by both endocrine and neural components that ultimately influence the energy intake and expenditure. Because of this complex regulatory mechanism even a small imbalance between the intake and expenditure will ultimately have large effects on body weight. Alterations in stable weight by forced overfeeding or food deprivation includes physiologic changes that resist these perturbations: with weight loss, appetite increases and energy expenditure falls, with overfeeding, the appetite falls and energy expenditure increases. This compensatory mechanism frequently fails, however permitting obesity to develop when food is abundant and physical activity is limited. A major regulator of these adaptive responses is the adipocytes –

derived hormone leptin which acts through brain circuits (predominantly in the hypothalamus) to influence appetite, energy expenditure and neuroendocrine functions.

There are several circuits within the hypothalamus that contribute to its role in integrating appetite, the melanocortin pathway being the most understood. The circuit begins with an area of the hypothalamus, the arcuate nucleus that has outputs to the lateral hypothalamus and ventromedial hypothalamus, the brains feeding and satiety centers, respectively. The arcuate nucleus contains two distinct groups of neurons. The first group coexpresses neuropeptide Y (NPY) and agouti–related peptide (AGRP) and has stimulatory inputs to the LH and inhibitory inputs to the ventromedial hypothalamus. The second group coexpresses pro-opiomelanocortin (POMC) and cocaine and amphetamine regulated transcript (CART). It has stimulatory inputs to the ventromedial hypothalamus and inhibitory inputs to the LH. Consequently, NPY/AGRP neurons stimulate feeding and inhibit satiety, while POMC/CART neurons stimulate satiety and inhibit feeding. Both groups of arcuate nucleus neurons are regulated in part by leptin. Leptin inhibits the NPY/AGRP group while stimulating the POMC/CART group. Thus a deficiency in leptin signaling, either via leptin deficiency or leptin resistance, leads to overfeeding or may account for some genetic and acquired forms of obesity. Peripheral organs participating in regulation of food intake are stomach, gut, pancreas and adipose tissue. Stomach and duodenum secrete gherlin which is orexigenic. Pancreas secretes insulin; adipose tissue secretes leptin. Both are anorexigenic.

Obesity has a profound effect on the physiology of breathing [(14)]. Obesity can lead to pulmonary compromise in a number of ways, including decrease in respiratory compliance due to mechanical factors such as increased weight on the thoracic cage and abdomen as well as changes in lung compliance. Airway obstruction at low lung volumes may also stimulate flow receptors to increase the sensation of breathlessness [(15, 16)]. Respiratory muscle weakness and fatigue may lead to dyspnea through an increase in effort (sensed in the somatosensory cortex) and chemoreceptor activity responding to increasing CO_2 production and hydrogen ion release.

Most of these respiratory function abnormalities seen in obesity are due to the mechanical load of adipose tissue on the chest wall and resultant deconditioning, it would be expected that obesity reduction in would lead to improvement in many of these physiological derangements.

III. Material And Methods

Material

The present study was conducted at the centre of VLCC, Aundh, Pune, using portable Helios digital spirometer machine from the Department of Physiology, Dr. D. Y. Patil Medical College, Pimpri, Pune. The study group included 90 obese individuals (both male and female) aged between 18-30 years enrolled for weight reduction program at above mentioned center.

Inclusion Criteria

1. Age: 18-30 years
2. Both males and females
3. Body Mass Index (BMI) ≥ 25

Exclusion Criteria

1. Morbid condition related to any systemic disease.
2. Smokers and alcoholics
3. Individuals suffer from nasal allergy or Atopy.
4. Obstructive sleep apnea syndrome.
5. Inability to perform the tests adequately.

They were categorized in three groups depending on target weight loss

a) Group1-Individuals with target weight loss >10kgs.
b) Group2-Individuals with target weight loss between 5kg to 10 kg
c) Group3-Individuals with target weight loss <5 kg .

Anthropometrical measurements were taken along with preliminary clinical examination to exclude anysystemic disorder.

Anthropometry

Age: Age was calculated from date of birth of subjects and controls.

Body Weight: A weighing scale was used to measure body weight with an accuracy of $\pm$ 100 grams (0.1 kgs). Subjects were weighed without their shoes and with light summer clothing.

Height: Standing body weight was measured without shoes with the use of height stand with shoulders in relaxed position and arms hanging freely.

Preliminary Clinical Examination

Include history taking, general examination and systemic examination of cardiovascular and respiratory system.

Recording Peak Expiratory Flow Rate

The questionnaires were filled up and the relevant data, name, age, sex, height, weight, occupation, smoker or nonsmoker, lab temperature was entered into the computer. All the subjects were made familiar with the instrument and procedure.The subject was connected to the mouthpiece and was asked to breathe in order to familiarize himself with the equipment.During the tests the subject was adequately encouraged to perform at their optimum level and also a nose clip was applied during the entire maneuver. One deep breath with full expiratory effort was performed through mouth piece. Three such readings were taken and best reading amongst all readings was chosen to be the final peak expiratory flow rate reading.

The outcome of peak expiratory flow rate was presented as a mean $\pm$ SD. The peak expiratory flow rate values within a group pre weight loss and post weight loss groups were compared by applying student 't' test and p value of less than 0.05 was considered as significant.

IV. Observations and Result

Table 1: Anthropometric Parameters Of The Study Groups

Parameters	GROUP 1 (n=22) Mean	GROUP 2 (n=30)	GROUP 3 (n=30)	p value
Age(years)	26.71± 2.81	26.90± 2.81	27.00± 2.39	0.93
Weight (kgs)	79.57± 11.33	75.38± 6.53	67.59± 5.26	0.00*
Height (cm)	160.05± 11.36	162.86± 5.26	159.93± 5.66	0.25

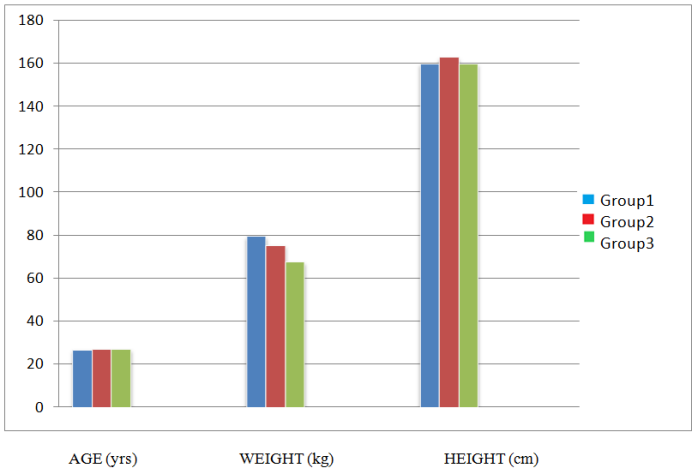
Results are expressed as mean ± SEM for normally distributed variables or as median inter quintile range when data was not normally distributed.

p* < 0.05

p** < 0.001

p*** < 0.0001

Table 1 shows ages and heights of all three groups were comparable. (p>0.05)Whereas, weights are significantly different. (p<0.05)



BAR DIAGRAM 1: SHOWING MEAN AGE (yrs), HEIGHT (cm) AND WEIGHT (kg)

Table 2: Peak Expiratory Flow Rate in All Study Groups Before And After Weight Loss.

PARAM-ETERS	GROUP 1		GROUP2		GROUP3	
	PRE	**POST**	**PRE**	**POST**	**PRE**	**POST**
PEFR	5.89±2.04	6.86±1.51**	8.44±1.20	8.77±1.07*	7.69±1.28	8.02±1.10**

p* < 0.05 statistically significant.

p** < 0.001 statistically highly significant.
p*** < 0.0001 statistically highly significant

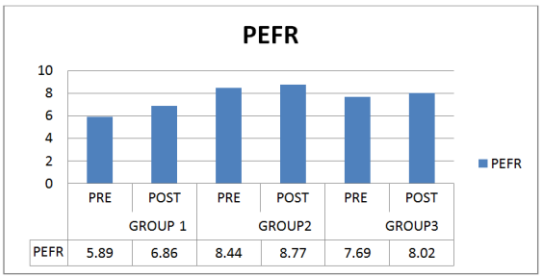
The comparison of peak expiratory flow rate pre and post weight loss was done with paired t-test in all study groups.

In Table 2,

Group 1 shows highly significant gain in PEFR (p=0.001)

Group 2 shows significant gain in PEFR (0.003).

In Group 3 shows highly significant gain in PEFR (p=0.0003).



BAR DIAGRAM 2: Peak Expiratory Flow Rate in All Groups

Table 3: Comparison of Peak Expiratory Flow Rate within All Study Groups before and After Weight Loss

PARAMETERS	GROUP 1	GROUP 2	GROUP 3	ANOVA
PEFR	**5.89±2.04**	**8.44±1.20**	**7.69±1.28**	**0.00***
PEFR POST	**6.86±1.51**	**8.77±1.07**	**8.02±1.10**	**0.00***

Table 3 shows one way analysis of variance which was conducted to explore the impact of weight loss on Peak expiratory flow rate. There was statistically difference at p level in all the groups**.**

V. Discussion

A significant improvement in the PEF values with weight loss was found. All three groups showed highly significant gain in Peak expiratory flow rate after weight loss**.** As there is diurnal variation in peak expiratory flow rate, all readings were taken at the same time of day every time. Though the mechanism of improvement in Peak expiratory flow rate is not clear it may be due to decrease in airway obstruction and improved ventilatory efforts after weight loss. PEFR depends on expiratory efforts exerted during forceful expiration as well as status of upper airways. PEFR reflects mainly the caliber of the bronchi and larger bronchioles, which are subjected to reflex bronchoconstriction. There is a strong association between forced expiratory flow rates and small airway function[(17)].These rates are reduced in obese individuals without any evidence of obstructive airway disease [(18)].This can be because of increased pulmonary blood volume in obese persons leading to congestion of bronchial vessels in the airway submucosa, thickening of airway wall and decrease in airway size[(19)].In these people presence of very low density lipoproteins can release histamine which is an effective mediator of vascular permeability and smooth muscle contraction. In addition to this airway resistance tends to increase the airway size at high pulmonary volumes and to reduce it at low pulmonary volume when the elastic recoil pressure diminishes [(20)]. It can be because of other mechanisms linking weight loss to the decrease in bronchial obstruction and hyperreactivity. WaddenTA, Considine RV,Foster GD,et al showed that Serum leptin levels changes after weight loss [(21)].Leptin may have proinflammmatory effects in the airways and may affect bronchial activity[(22)] .Gene polymorphism may alter adrenergic receptor responsiveness in obesity[(23)]. On comparison amongst these groups it was found that there was statistically significant difference in Peak Expiratory Flow Rate before weight loss as well as after weight loss. Thus it is clear from this study that weight has influence on peak expiratory flow rate and that weight loss has positive repercussion on it.

VI. Conclusion

This study indicates that the body weight of an individual has an effect on PEFR. It was seen that in all three groups there was significant improvement in PEFR after weight loss. Though the reasons for this difference are not clearly known but altered mechanical muscular activity due to adiposity, altered airway caliber and increase respiratory resistance along with remodeling of respiratory passage due to circulating inflammatory mediators may be responsible for less PEFR in obese and overweight individuals. Weight loss

may have reversed some of above changes and responsible for gain in peak expiratory flow rate. This can be implicated while treating respiratory diseases so that recovery from symptoms can be fastened.

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