Research Article

Effect of Body Mass Index on Cardio-Respiratory Parameters in Healthy Volunteers of Western Rajasthan

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Abstract: Obesity is the one factor which affects cardiac as well as pulmonary function. So our study is to explore Cardiac and Pulmonary parameters among normal weight, overweight and obese subjects. Total 152 subjects were selected randomly for study between age group 15 to 45 years. They were divided into three groups on the basis of their Body Mass Index (BMI). Group A, BMI-18.6 to 24.9Kg/m² (normal weight), Group B, BMI-25 to 29.9Kg/m² (overweight) and group C, BMI >30Kg/m² (obese). After recording of anthropometric measurements all the subjects underwent measurement of the resting heart rate, blood pressure and following spirometric values, Forced vital capacity (FVC) in liters, Forced expiratory volume in first second (FEV_1) in liters, Peak expiratory flow rate (PEFR) in liters/sec, Forced expiratory flow during the middle half of the FVC ($FEF_{25-75\%}$)liters/sec, $FEF_{25\%}$ ($Vmax_{25\%}$)liters/sec, $FEF_{50\%}$($Vmax_{50\%}$)liters/sec, $FEF_{75\%}$($Vmax_{75\%}$)liters/sec and FEV_1/FVC%. Our results show significant increase in Resting Heart Rate (RHR), Resting Blood Pressure (RBP) with increase in BMI. Spiromertic values FEV1/FVC% ratio and $FEF_{75\%}$ were significantly changed with change in BMI, but not any significant difference was found in Pulse Pressure, Mean Arterial Pressure and FVC, FEV1, PEFR, FEF_{25-75}, $FEF_{25\%}$, $FEF_{50\%}$, $FEF_{75\%}$, in different BMI groups. A significant increase in RHR, SBP and DBP with increase in BMI could point towards an altered autonomic balance with increase in body weight. Obesity does not have effect on the spirometry parameters among healthy nonsmokers, except FEV1/FVC% and $FEF_{75\%}$. We recommend searching for alternative diagnosis in case of findings abnormal spirometry tests results in obese.

Keywords: BMI, Obesity, Blood Pressure

INTRODUCTION

The autonomic dysfunction associated with obesity could lead to changes in resting heart rate (RHR) and arterial blood pressure [1]. Quantification of RHR and RBP can give an index of the load imposed on the heart and the imbalance state between sympathetic and parasympathetic activity [2]. RHR has been postulated as a screening index for cardiovascular risk and it has been supported by studies reporting its relationship to mortality [3, 4].

Obesity and pulmonary function have a historical association [5]. Obesity can cause different deleterious effects to respiratory function that include respiratory mechanics alteration, decrease in respiratory muscle strength and endurance, decrease in pulmonary gas exchange, lower control of breathing, and limitations in the pulmonary function tests and exercise capacity [6]. These changes in lung function occur due to the extra adipose tissue in the chest wall and abdominal cavity that compress the thoracic cage, diaphragm and lungs. The consequences are: decrease in displacement of the diaphragm, decrease in compliance of lung and chest wall and an increase in elastic recoil that result in a decrease in lung volumes and an overload of inspiratory muscles [6, 7]. These changes are found to be worsened by an increase in the BMI [8].

Since morbid obesity is always found to be in association with various alterations in pulmonary functions, it becomes necessary to assess the respiratory function of obese individuals [6]. Therefore, the objective of this study was, by comparing lung functions in normal weight, overweight and obese subjects, to evaluate the impact of body weight on the pulmonary function of healthy subjects with no history of pulmonary disease along with resting cardiac functions like resting Heart rate, Resting Blood Pressure

(systolic and Diastolic), Pulse Pressure and Mean Arterial Pressure.

Pulmonary function tests (PFT) values are used by most of the researchers for the investigation of the effect of obesity on the respiratory system. The factors usually affecting the values of pulmonary function tests include age, gender, height, race or ethnic origin and possibly obesity. As the individual gets older age, the lung volumes and capacities become smaller [9, 10].

This study is limited to spirometry part of PFT because spirometry tests are considered to be the initial screening tool for the assessment of pulmonary diseases. They are most widely used, easy to conduct by the use of equipment available in all pulmonary functions laboratories [10].

So the present study was aimed to investigate the correlation of RHR, resting blood pressure and Spirometric Parameters with body mass index (BMI)

MATERIALS AND METHODS
This is a cross sectional study conducted at Dr S N Medical College, Jodhpur, Rajasthan. After approval of The Institutional Ethical Committee we selected the subjects randomly from the male and female population (aged 15 to 45 years) of healthy volunteers. Weight was recorded in kgs using a calibrated weighing machine scale with a capacity of 120 kg, while subject wearing light cloths. Height was measured in centimeters (cm) bare foot against a wall with the help of a measuring tape

Subjects were lifetime nonsmoker, had no history of respiratory diseases such as pulmonary tuberculosis or asthma and not having history of cardiac or thoracic surgery or features suggestive of cardiac or lung disease or evidence of chest deformities or serious medical conditions, and not having worked in environments with a high concentration of dust or pollution.

All subjects were explained about the procedure to be undertaken and informed written consent was obtained. These subjects were given a proforma to fill up certain details like their dietary habits, extent of physical activity and family history. They were asked to avoid food, tea, coffee, nicotine at least two hour prior to testing.

Heart Rate and Blood Pressure were measured after complete rest of 15 minutes in lying position. Heart Rate counting was done by counting the pulse rate three times and average was taken to diminish manual error. Resting Blood Pressure was measured by Sphygmomanometer by Auscultatory method. The subjects underwent the spirometry test in the standing position, wearing a nose clip. Uniformity of spirometry test was assured by using the same device brand for all the subjects. Pulmonary functions were measured with the help of computerised spirometer **"Medicaid spiro excel"** (portable). Pulmonary parameters recorded and studied were: Forced vital capacity (FVC) in liters ,Forced expiratory volume in first second (FEV_1) in liters, Peak expiratory flow rate (PEFR) in liters/sec, Forced expiratory flow during the middle half of the FVC ($FEF_{25-75\%}$) liters/sec, $FEF_{25\%}$ liters/sec, $FEF_{50\%}$ liters/sec , $FEF_{75\%}$ liters/sec , FEV_1 /FVC% . Details of age, sex, height and weight were recorded and entered in machine.

Procedure for recording
The subject was asked to breathe in and out normally. Then he was asked to take a deep breath and fill his lungs to maximum possible and then exhale into the mouth piece as quickly as possible. The subject was asked to make three attempts and the best of them was selected.

Statistical analysis
The statistical analysis was performed using the GraphPad InStat3 software. Descriptive statistics were calculated for the total study sample, for different age groups and Body Mass Index (BMI) groups using means and standard deviations. The variables were expressed as the means and standard deviations, and p-value less than 5% was considered statistically significant. Independent samples test was used to compare the spirometry results of normal weight, overweight and obese subjects.

RESULTS AND DISCUSSION

Table 1: Anthropometric parameters in normal weight, overweight and obese subjects

Parameters	Normal weight (n=70)	Overweight (n=44)	Obese (n=38)
Age	26.2143 ± 7.7195	30.7045 ± 9.9500	31.7632 ± 9.0267
Height	164.5 ± 7.1864	166.295 ± 9.4119	164.555 ± 6.6523
Weight	56.4286 ± 6.3578	73.8636 ± 9.3624	85.6316 ± 7.7752
BMI	20.6571 ± 1.4831	26.6136 ± 1.0165	31.5263 ± 1.3504

Table 2: Cardiac parameters of normal weight, overweight and obese subjects

	Normal weight (n=70)	Overweight (n=44)	Obese (n=38)	p-value
RHR	72.4429 ± 8.4418	74.5909 ± 11.7776	80.4211±12.1802* #	<0.001
SBP	116.029 ± 13.3384	117.5 ± 10.0337	123.842 ± 11.4409* #	<0.05
DBP	69.4286 ± 8.4245	73.8182 ± 8.2045˙	77.3158 ± 7.8949*	<0.001
PP	46.6 ± 9.1768	43.6818 ± 6.8464	46.5263 ± 8.8676	NS
MAP	69.7427 ± 9.9841	68.2882 ± 7.0600	70.9037 ± 19.9696	NS

p-value <.05 =significant

▪ Normal weight and Overweight intergroup comparison was significant by post hoc test (Tukey Kramer Multiple Comparison Test)

* Normal weight and Obese intergroup comparison was significant by post hoc test (Tukey Kramer Multiple Comparison Test)

Overweight and Obese intergroup comparison was significant by post hoc test (Tukey Kramer Multiple Comparison Test)

Table 3: Spirometric parameters in normal weight, overweight and obese groups

Parameters	Normal weight (n=70)	Overweight (n=44)	Obese (n=38)	p-value
FVC	2.9795 ± 1.1918	2.825 ± 0.6963	2.5984 ±0.4064	NS
FEV1	2.4708 ± 0.7546	2.5336 ± 0.5998	2.3884 ± 0.3619	NS
FEV1/FVC%	84.2787 ± 18.0556	89.953 ± 5.3437	92.1092±5.0275*	<0.05
PEFR	7.2715 ± 1.9425	7.4511 ± 2.3599	6.8271 ± 1.4661	NS
$FEF_{25-75\%}$	4.5984 ± 1.6414	4.5086 ± 1.5551	4.4442 ± 1.0741	NS
$Vmax_{25\%}$	6.9645 ± 1.9968	7.2225 ± 2.2556	6.7268 ± 1.5277	NS
$Vmax_{50\%}$	5.0085 ± 1.8833	5.1225 ± 1.7837	5.1110 ± 1.2088	NS
$Vmax_{75\%}$	2.8792 ± 1.2591	2.3720 ± 1.0995	2.4194 ± 0.8333	<0.05

p-value <.05 =significant

*Normal weight and obese intergroup comparison was significant by post hoc test (Tukey Kramer Multiple Comparison Test)
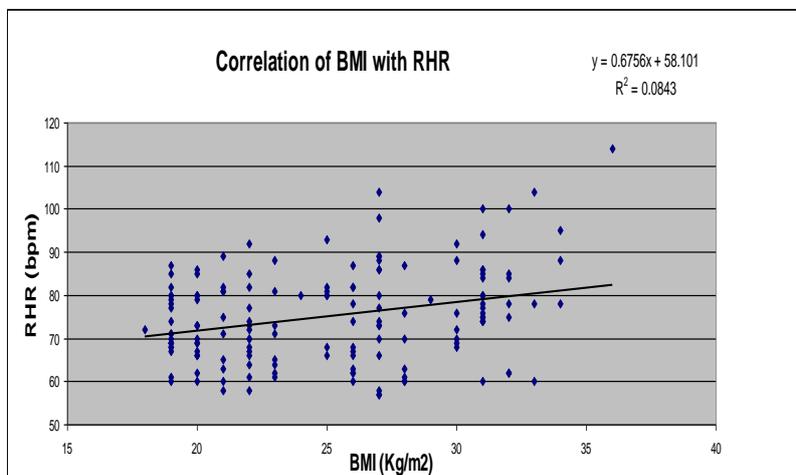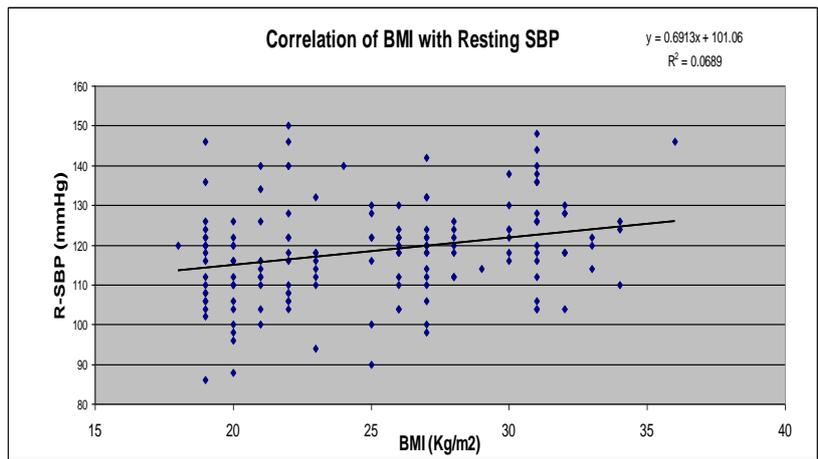

Fig.1: Correlation of BMI with RHR

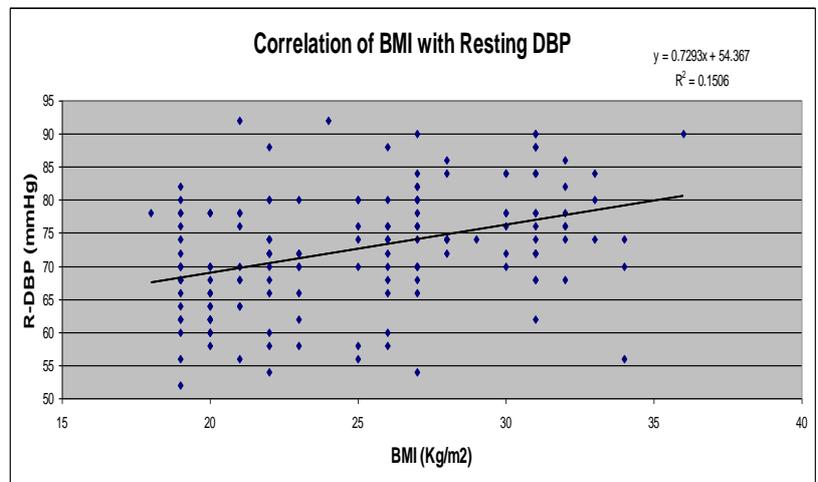**Fig. 2: Correlation of BMI with resting SBP**

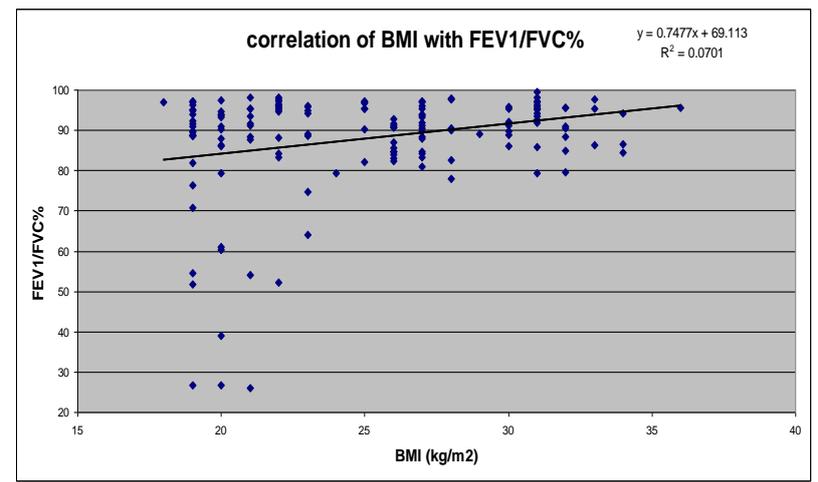**Fig. 3: Correlation of BMI with resting DBP**

Fig. 3: Correlation of BMI with FEV1/FVC%

Obesity is known to cause autonomic dysfunction and RHR and Blood pressure are dependent on autonomic system [11]. Hence obesity can lead to change in RHR and arterial blood pressure.

The main results of our study showed that there was significant increase in resting Heart Rate (RHR), Systolic (SBP) and Diastolic Blood Pressure (DBP) in normal weight Overweight and Obese Groups respectively. There were no significant differences in FVC, FEV1, PEFR, $FEF_{25-75\%}$, $FEF_{25\%}$ ($Vmax_{25\%}$), $FEF_{50\%}$ ($Vmax_{50\%}$) among normal weight, overweight and obese subjects, except PEFR and $FEF_{75\%}$ ($Vmax_{75\%}$).

An elevated Heart Rate (HR) is a warning about an increased risk of cardiovascular dysfunction and an increase in heart rate by 10 beats per minute is said to be associated with an increase in the risk of cardiac death by at least 20%, which is similar to the risk observed with an increase in systolic blood pressure by 10 mmHg [12].

The heart rate is an integrated index of autonomic cardiovascular function and elevated heart rate values indicate adrenergic overdrive that leads to / or worsening ischemia with risk of acute coronary syndromes (ACS), fatal or non-fatal arrhythmias or heart failure. Secondly, the elevated heart rate exerts mechanical effects on the cardiac vasculature that leads to increased shear stress, impaired arterial compliance and favours atherosclerotic vascular lesions development [13, 14].

We observed that there was high RHR in Overweight and obese group as compared to normal weight group. It was also found that there was a positive and significant correlation of RHR with obesity parameters like BMI. Studies done by Dimkpa *et al.* [15] and Grassi *et al.* [16] also observed a significant increase in RHR with increase in body weight, which were similar with our study.

The resting heart rate is influenced by several factors that include genetic characteristics, anthropometrics, body posture, age, gender, hormonal and emotional factors, and physical fitness level. Different mechanisms are found to be involved that causes cardiovascular complications in obese individuals due to the increase in heart rate. Higher heart rate can directly increase the consumption of myocardial oxygen and induces break up of arterial wall elastic fibers. Obesity and cardiac autonomic nervous system are related. Elevated heart rate indicates an increased risk of cardiovascular dysfunction [17]. NHANES study has reported that a heart rate of more than 80 bpm indicates a greater risk of cardiovascular complication [18]. An increase in body weight has been found to be associated with an increased RHR that results in increased in sympathetic and simultaneously decreased parasympathetic activity. In converse, heart rate decreases during weight reduction [19]. Increased parasympathetic activation due to weight reduction is beneficial for the individuals with obesity [17].

Masaki KH *et al.* reported the close relationship between BMI and basal systolic and diastolic blood pressures [20]. Similar results have been reported in China by Huang *et al.* [21].

The PP and height relation was augmented comparably in boys and girls who were obese (based on BMI) [22].

Many studies have reported that such relationship of obesity and pulmonary functions showed heterogeneous results. The effects of obesity on spirometric values are not consistent in all studies. Some have reported no effects and while some other studies have shown positive effects. This discrepancy can be explained by the wide variations in ethnicity of different population or may be a result of methodological differences in the conducted studies [10].

Author Costa D *et al.* [6] reported the relationship between spirometric tests and obesity produced results similar to those of our study. Their study included 20 obese young women with a BMI of 35-49.99 kg/m^2 who were sedentary, non-smokers and without lung disease and 20 non-obese control young women with same inclusion criteria BMI between 18.5 and 24.99 kg/m^2. They found no significant differences between the obese group and the non-obese group with respect to the age, vital capacity (VC), tidal volume (TV), FVC, and FEV1. The obese group had a greater inspiratory reserve volume (IRV), a lower expiratory reserve volume (ERV), and lower maximal voluntary ventilation (MVV) than the non-obese group [1].

Ray CS *et al.* [23] studied spirometry, lung volume measurement by nitrogen washout, and single-breath diffusing capacity for carbon monoxide (DLCO) on 43 massive obese, observed that vital capacity, total lung capacity, and maximal voluntary ventilation change only with extreme obesity. They concluded that abnormal pulmonary function test value should be considered as caused by intrinsic lung disease and not by obesity, except in those with extreme obesity.

Li *et al.* [24] had studied 64 obese adolescents and found decreased FVC only in morbidly obese adolescents (BMI $\geq$ 45 kg/m2). They observed that only three of the 64 patients had evidence of pulmonary obstruction with BMI > 34 kg/m^2. The same has been demonstrated with mild degrees of obesity, which should result in less reduction in FEV1 and FVC values, as observed in the this study.

Some authors have suggested that obesity may promote air trapping, which impairs adequate pulmonary ventilation through the reduction of pulmonary volumes [6]. Ladosky *et al.* comparing a group of obese and non-obese patients, suggested that the reduction of the ERV may be a consequence of air trapping caused by obesity and leading to a reduction in the MVV [25]. This reduction in the ERV can be attributed to the obstruction of small airways and a consequent reduction in gas exchange.

Here with comparison of other studies, it is clear that obesity affecting the pulmonary functions only in obese having BMI value more than 34 Kg/m^2.

Fat distribution pattern is more representative when compared only to the BMI. Abdominal obesity is often correlated with reduced FVC and FEV1 [26]. Faria AG *et al*. had reported that obese adolescents had increased concentrations of abdominal fat, a fact observed during the measurement of waist circumference [27].

So we can say that, our study had the limitation of using BMI as an indictor of obesity. BMI is a global measure of body mass that includes both fat and lean mass and takes no account of differences in fat distribution. If the reduction of lung volumes in obesity is due to a direct mechanical effect on lung volumes, then the distribution of body fat should modify the relationship between BMI and lung volumes. 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 less likely to have any direct mechanical effect on the lungs. However, the standard classification of obesity uses BMI as a reflection of obesity and this classification is used globally by WHO and other related health organizations. It is considered to be the gold standard at the present time.

In the present study there was a greater change in RHR in overweight and obese group compared to normal weight group. This could be explained on the basis of a relatively higher sympathetic tone in the obese group than NW or the parasympathetic tone was comparatively less in the obese compared to the NW.

The present study showed hat there is a significant direct correlation between obesity and RHR, with the obese group exhibiting a significantly faster RHR compared to normal weight group. This faster RHR in obese individuals could contribute to various cardiovascular problems in later life. Thus it can be concluded from the results of the present study that there was altered cardiac autonomic activity in obese individuals. Obese group showed a significant reduction of parasympathetic activity and a significant increase in sympathetic activity. This shows imbalance in the autonomic neural activities of the heart. Hence there is need to prevent obesity early in life to avoid life threatening cardiovascular consequences in advancing age. Thus early interventional programme like weight reduction, life style changes and physical exercises can be advised to reduce the chances of subsequent cardiac problems.

CONCLUSION

In conclusion, obesity does not have direct effect on the spirometry tests results among healthy non-smoking subjects and if there is any effect, it should be explained by alternative diagnosis or underlying respiratory diseases. We strongly recommend conducting larger study including all pulmonary functions tests variables (spirometry tests and other lung volumes). It will interesting to use other indices of obesity like abdominal girth, subscapular skin fold thickness, and the ratio of abdominal girth to hip breadth as reflection of obesity instead of BMI and find if such results will differ in comparison to the results of using BMI. Based on our result, we highly recommend to physicians using spirometry test in their practice to search for alternative diagnosis in case of findings abnormal spirometry tests results among obese individuals as these abnormal findings should not attributed to obesity.

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