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Study of Cardio Respiratory Parameters in First Year Medical Students with Special Reference to Anthropometric Indices

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Abstract:

Rationale: The price we are paying for an affluent and developed society is a sedentary life style and faulty dietary habits which results in an imbalance between energy intake and energy expenditure, which in turns lead to obesity. Increasing prevalence of obesity in the adolescents worldwide is now a major concern. Aim: To study, that there is statistically significant increase in heart rate, systolic blood pressure and diastolic blood pressure in overweight subjects when compared to normal weight subjects. Similarly, there is a statistically significant decrease in FVC and PEFR in overweight when compared to normal weight subjects. Methods: Study will be carried out at Government medical college, Nagpur. Subjects will be selected from 18-22 years age group. Estimated total sample size will be 200 subjects. Cardio-respiratory parameters such as heart rate, systolic blood pressure, diastolic blood pressure, forced vital capacity and peak expiratory flow rate will be assessed. Results: all the cardiac parameters i.e. heart rate, systolic blood pressure and diastolic blood pressure were significantly increased in overweight subjects when compared with controls. While, respiratory parameters i.e. FVC (forced vital capacity) and PEFR (peak expiratory flow rate) were reduced in overweight subjects when compared with controls. Conclusion: There is a positive correlation between increase in body mass index (BMI) and change in heart rate, systolic blood pressure and diastolic blood pressure.

•There is a negative correlation between increase in body mass index (BMI) and change in forced vital capacity (FVC) and peak expiratory flow rate (PEFR).

1. Introduction

Measuring height, weight, and body mass index (BMI) gives significant information on the nutritional and health status of individuals. BMI is positively and independently associated with morbidity and mortality from hypertension, cardiovascular diseases, diabetes mellitus, asthma, and other chronic diseases.^{i,ii}

Overweight and obesity represent a rapidly growing threat to the healthy populations, even in developing countries like India³.

Overweight and obesity are fifth leading risks for global deaths³. Worldwide, obesity has more than doubled since 1980. In 2008, more than 1.4 billion adults, 20 years and older, were overweight. Of these, over 200 million men and nearly 300 million women were obese.

In India, 1.3 per cent males and 2.5 per cent females aged more than 20 years were obese in the year 2008ⁱⁱⁱ.

Obesity is perhaps the most prevalent form of malnutrition. As a chronic disease, prevalent in both developed and developing countries, and affecting children as well as adults, it is now so common that it is replacing the more traditional public health concerns including undernutrition. It is one of the most significant contributors to ill health. For industrialized countries, it has been suggested that such increase in body weight has been caused primarily by reduced levels of physical activity, rather than by changes in food intake or by other factors. It is extremely difficult to assess the size of the problem and compare the prevalence rates in different countries as no exact figures are available and also because the definitions of obesity are not standardizedⁱⁱⁱ.

In developing countries like India, transport facilities, medical care and food habits, educational status, and family income have dramatically improved, which, along with easy access to city and television watching, result in unwanted changes in lifestyle. These have eventually led to a significant increase in body mass index (BMI) as well as abdominal obesity in both sexes as compared to a similar study conducted in the year 1989. The prevalence of overweight rose from 2 to 17.1%. The changing lifestyle of the rural dwellers was found to be a contributory factor for the rising rates of obesity and associated metabolic diseases such as diabetes^{iv}.

Anthropometric measurements are universally applicable, simple, inexpensive and non-invasive techniques to measure obesity, but still are underutilized tools for guiding public policy.

Body mass index (BMI) is calculated as the weight in kilograms divided by the square of the height in meters ($BMI = \text{weight (kg)}/\text{height (m)}^2$). The World Health Organization (WHO) classified obesity using BMI cut-off values of 25 and 30 kg/m². Body mass index (BMI) of 18 to 24.9 kg/m² is considered normal weight, a BMI of 25.0-29.9 kg/m² is considered overweight and a BMI of 30 kg/m² or higher is considered obesity^v.

Obesity has been associated with many health consequences, including but not limited to diabetes, hypertension, hyperlipidemia, ischemic heart diseases, obstructive sleep apnoea, stroke, premature death, osteoporosis and a reduction of the overall quality of life^{vi}. In 2013, the American Medical Association classified obesity as a disease^{vii}.

If everyone is at its optimal weight, there would be 25% less coronary artery disease and 35% less congestive heart failure and stroke. If body mass index of the population becomes normal, this would produce a total reduction in mortality of 15%, corresponding to 3 years added life expectancy^{viii}.

Increasing prevalence of obesity in the adolescents worldwide is now a major concern. Current dramatic rise of obesity among adolescent portends a future wave of increasing cardiovascular diseases as these overweight youth reach the adult years.

A variety of adaptations in cardio-respiratory structure and function occur in the individual as adipose tissue accumulates in excess amounts even in the absence of co-morbidities. Hence, obesity may affect the heart and lungs through its influence on known risk factors such as dyslipidemia, hypertension, glucose intolerance, inflammatory markers, obstructive sleep apnoea, hypoventilation and prothrombotic state. Studies of conventional respiratory function tests in overweight subjects have generally shown relatively minor effects unless obesity is extreme.

The the present study aimed to explore the association between anthropometric indices like weight, height, body mass index (BMI) and cardio-respiratory parameters like heart rate, systolic blood pressure, diastolic blood pressure, and forced vital capacity (FVC) and peak expiratory flow rate (PEFR). Although present study is by no means exhaustive, it does provides a glimpse into the variety of adaptations/alterations in cardio-respiratory function that occur as excessive adipose tissue accumulates, even in absence of overt diseases.

2. Aims & Objectives

To study cardiac parameters like Heart rate, Systolic blood pressure, diastolic blood pressure, mean arterial pressure & pulse pressure and respiratory parameters like Forced vital capacity & Peak expiratory flow rate and its correlation with anthropometric indices such as Height, Weight & Body mass index in first year Medical students of 18-22 years of age group.

3. Methods

The the present study was conducted in the department of physiology at medical college. Approval of the institutional ethics committee was taken prior to the start of the study. A sample size of 200 was estimated from a study population of first year medical and dental students of the institute. Subjects were selected by random sampling, and informed written consent was taken for participation in the study after detailed explanation of the purpose and method of conducting this study.

The study was undertaken to analyze the differences in certain cardio-respiratory parameters in healthy normal controls & overweight subjects in the age group 18-22 years.

Study Subjects were divided into two groups.

3.1. Selection Criteria

1. Group A (Controls) :

Healthy normal subject having body mass index 18.50-24.99 kg/m²

2. Group B (Overweight):

Overweight subjects of body mass index 25.00-29.99 kg/m²

3.2. Inclusion Criteria : (Suggested by Taussig et al.)^{ix}

1. Absence of chronic airway disease
2. No history of acute respiratory tract infection preceding three weeks.
3. No major respiratory disease or thoracic surgery in the past
4. No systemic disease influencing the respiratory system
5. Non-smoker

3.3. Exclusion Criteria

1. Those with the history of addiction to tobacco, alcohol, smoking.
2. Those having BMI ≥ 30 Kg/m² (obese subjects) and ≤ 18.5 Kg/m² (underweight subjects).
3. Those suffering from any other disease or complications.
4. Those having history of hypertension and diabetes in the past or present
5. Those suffering from any acute or chronic diseases or any other systemic illness that directly or indirectly affects cardio-respiratory parameters will be excluded.

In the the present study each subject was made familiar with the procedure to alleviate any fear or apprehension. The physical examination of all the subjects before the start of the procedure was done. The consent form was signed by the subjects before the procedure.

3.4. Data Was Collected Under Following Headings

1. Personal Information :

Includes name, age, sex, history of present and past illness if any, personal history and family history.

2. Anthropometric measurements :

Include height, weight and body mass index.

3. General examination :

Includes condition of the participant, temperature, pulse rate, respiratory rate, blood pressure, oedema feet and any other relevant findings.

4. Systemic examination:

It was carried to rule out any major diseases of cardiovascular system, respiratory system, alimentary system and central nervous system.

5. Pulmonary function test:

It was performed with electronic computerized spirometer RMS HELIOS 401 version.

3.5. Methodology Used for Measuring Anthropometric Indices and Certain Cardio-Respiratory Parameters Includes

3.5.1. Anthropometric Indices

a) Standing Height:

It was measured by making the subject stand against a wall on which measuring scale was inscribed. Standing height was measured as the subject stood erect with bare feet on flat floor with heels close together and arms hanging naturally at the side, heels, buttocks and occiput were touching the wall firmly. The external auditory meatus and the lower border of the orbit were in a plane parallel with the floor. The highest point of the vertex was marked on the wall by plastic ruler and then height was measured to nearest completed centimetres.

b) Weight :

Body Weight (kg) was recorded with human weighing machine in a standing position without shoes to the nearest 100 gm with minimal clothes.

c) Body mass index³:

Body mass index (Quetelet's index) was calculated, as given below–

BMI was calculated as–

$$\text{BMI} = \frac{\text{Weight in Kilograms}}{\text{Height in meter}^2}$$

3.5.2. Cardiac Parameters

a) Blood Pressure Measurement (mm of Hg):

The subjects were initially made to rest for 15 minutes. Blood pressure measurement was done in study subjects in sitting position. Based on the circumference of the subject's arm, a regular adult size cuff was chosen. The arm bare up to the shoulder and cuff was placed on the subject's right arm and blood pressure was first recorded by palpatory method and then confirmed by auscultatory method.

b) Pulse Rate (beats per min) :

Pulse rate was measured at the radial artery of the comfortably seated subject using index, middle and ring finger by the examiner. The index finger was used to obliterate the retrograde pulsations. Middle finger was used to assess the pulse rate. Ring finger was used to assess the force and tension of the pulse. The pulse rate was measured for one minute.

c) Pulse Pressure (mm of Hg):

Pulse Pressure (PP) = Systolic BP (SBP) – Diastolic BP (DBP)

d) Mean arterial Pressure (mm of Hg) :

Mean arterial pressure (MAP) = DBP + 1/3 (Pulse Pressure)

3.5.3. Respiratory Parameters

a) Pulmonary Function Test (PFT):

Subjects were explained about the whole procedure in detail and were motivated prior to the start of man oeuvre. The test was performed over 3 times. Test with best manoeuvre was selected.

The instrument used is RMS HELIOS 401 version, a electronic computerized spirometer (manufactured by Med. System Pvt. Ltd, Chandigarh). The instrument fulfils the criteria for performance and reproducibility laid down by American Thoracic Society (ATS).

The subjects were given standard instructions and detailed information with necessary demonstrations. The reading was taken in a comfortable upright sitting position in front of the apparatus.

Initially, following data was fed into the instrument:

Date: Room temperature:

Name: Age: in years

Sex: M/ F Height: in cm

Weight: in Kg Case No :

With the help of this data and standard regression equations in the software of the microprocessor, predicted values of pulmonary function parameters were calculated by instrument and were converted at BTPS (Body Temperature and Pressure Saturated) by the instrument itself.

Test Techniques:

Subject was asked to take a maximum inspiration and pinch nose, and then expire forcefully and completely in the mouthpiece of the instrument. The procedure was repeated for three consecutive times with adequate rest between each reading and the best one was noted.

Following are the parameters chosen for the study, which are more relevant and pertinent to the study:

1)FVC (lit)

2) PEFR (lit / sec)

FVC -Forced vital capacity or timed vital capacity is the volume of air that can be expired rapidly with a maximal expiratory effort after maximal inspiration.

Components of FVC are FEV1, FEV2 and FEV3.

FEV1 -It is the volume of air expired during the first second of FVC. It is most commonly used test for airway diseases.FEV1 is a flow rate. Its unit is L/sec.

FEV1% -It is the percentage of VC expired in one second. Normally FEV1% is 80% of FVC.

PEFR - Peak Expiratory Flow Rate (PEFR)

It is the maximum rate of air flow observed during a sudden forced expiration, from the position of full inspiration.

Normal value for adult male is 450-550 litres/minute.

Normal value for adult female is 350-450 litres/minute.

For each subject a printed sheet of predicted, pre(observed) and percentage predicted values of all respiratory function parameters were taken. The parameters can also be explained with the help of graphical representation in the form of flow/volume plot and volume /time plot.

3.6. Statistical Analysis

All the continuous variables were presented as Mean \pm standard deviation (SD). Categorical variables were expressed in actual numbers and percentages. continuous variables (age, PFT parameters) were compared between control and overweight subjects by performing unpaired t-test and also for comparing sex wise differences. Correlation coefficient (μ) was assessed to determine the nature and strength of correlation between BMI and cardio-respiratory parameters. Cardio-respiratory, PFT variables were compared between different age groups by performing one way ANOVA test. P value <0.05 was considered as statistical significant. Statistical software STATA version 13.1 was used for data analysis.

4. Results

200 study subjects have participated. They are divided into two groups (100 each) on the basis of body mass index.

These two groups are mentioned as below:

→ Group A (Controls): Healthy normal subjects having body mass index 18.50-24.99 kg/m²

→ Group B (Overweight): Overweight Subjects having body mass index 25.00-29.99 kg/m²

Study of cardio-respiratory parameters are done in all the study groups and compared between Group A and Group B. The results obtained in the study groups are tabulated as given below:

p value > 0.05 : Non-significant (NS)

<0.05 : significant (S)

<0.01 : significant (S)

<0.001 : highly significant (HS)

Age (Years)	Control	Overweight
18	44	38
19	48	45
20	08	17
21	00	00
22	00	00
TOTAL	100	100
Mean Age (years)	18.64 \pm 0.62	18.79 \pm 0.71

Table 1: Distribution of study subjects on the basis of Age (years) and comparison of the mean values of Age (years) between Control and Overweight.

Above table shows the mean values of age (years) in controls (18.64 ± 0.62) and overweight subjects (18.79 ± 0.71) which is found to be statistically non-significant. (t value= 1.59, p value = 0.12). Maximum number of study subjects are in the age group of 19 years. None is in the age group 21 and 22 years. Thus, mean age (years) is comparable in both controls and overweight subjects.

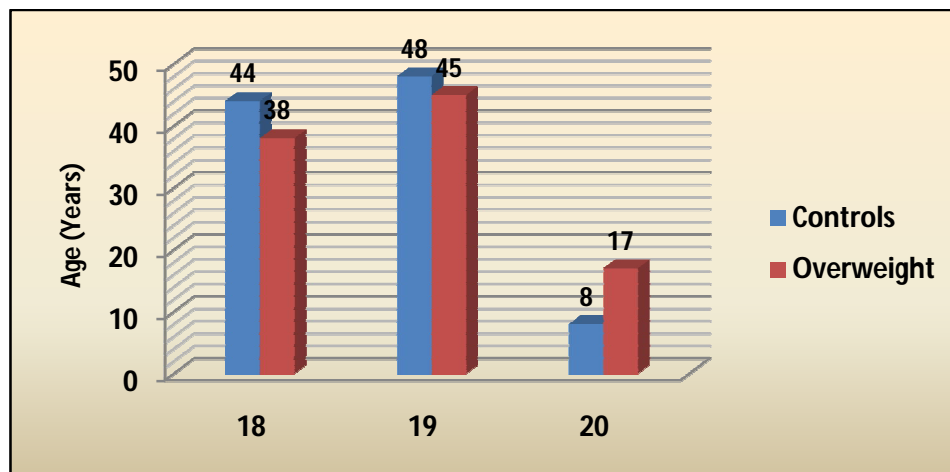


Figure 1: Age distribution

Sex	Control	Overweight
Male	59	62
Female	41	38
Total	100	100

Table 2: Distribution according to Sex*
 { * $\chi^2=0.17$ degree of freedom 1 }

The above table shows 59 % and 62 % of males are present in control and overweight group respectively. Whereas, 41% and 38 % of females are present in controls and overweight group respectively. Thus, males are more in number than females in both the groups which is found to be statistically non-significant (p=0.66).

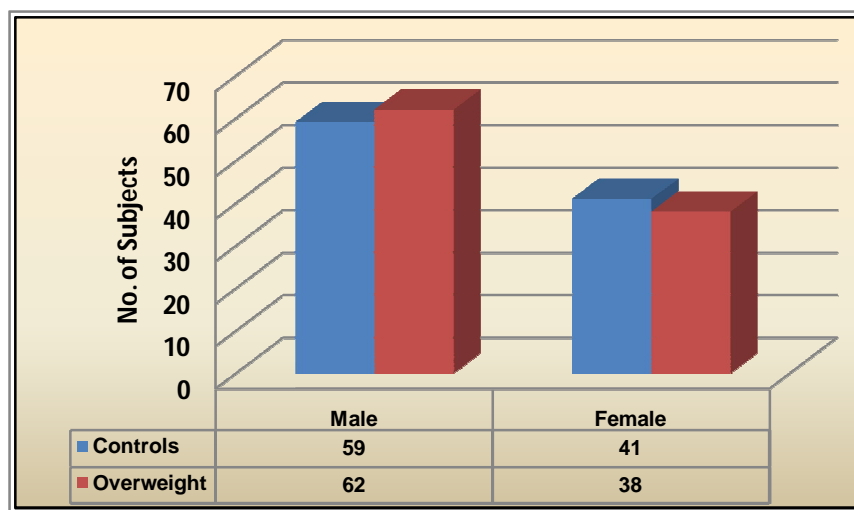


Figure 2: Sex distribution

Variable	Control	Overweight	t-value	p-value
Weight	55.82 ±8.26	65.82 ±6.57	9.47	<0.0001, HS
Height	162.83 ±9.13	159.09 ±7.38	2.17	<0.01,S
BMI	20.97 ±1.95	25.94 ±0.90	23.14	<0.0001, HS

Table 3: Distribution of study subjects according to anthropometric indices and comparison of the mean values of Weight (Kg), Height (cm) and Body mass index (Kg/m²) between control and overweight group.

The mean value ± standard deviation (Mean± SD) of BMI is found to be 25.94 ± 0.90 in overweight group and 20.97 ± 1.95 in controls. When unpaired t test is applied, it is found to be statistically highly significant (t value=23.14,p value < 0.0001).

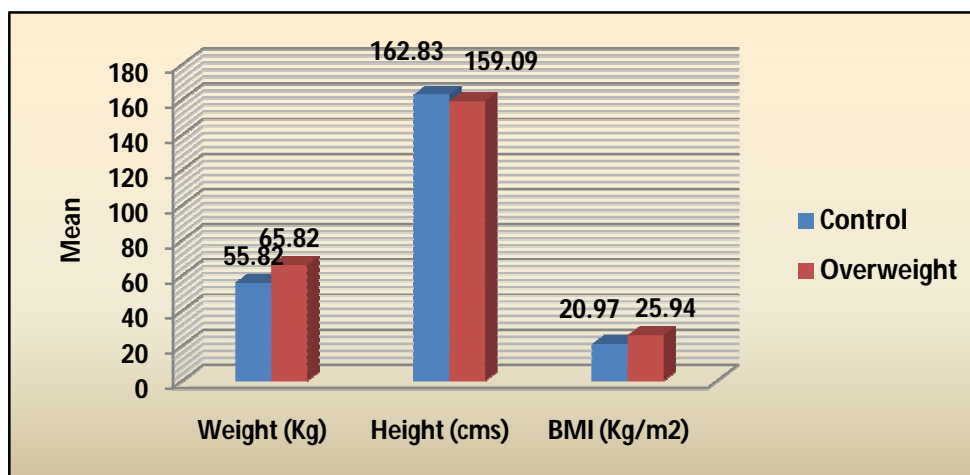


Figure 3: Anthropometric characteristics

Parameter	Control	Overweight	t-value	p-value
Heart rate	75.5± 5.04	84.79 ± 4.89	13.22	<0.0001, HS
SBP	116.82± 4.06	122.32 ±5.25	8.28	<0.0001, HS
DBP	76.32± 3.95	81.68 ± 3.65	9.97	<0.0001, HS
MAP	89.82 ±3.03	95.22± 3.23	12.19	<0.0001, HS
PP	40.50 ± 5.48	40.64 ±5.66	0.17	>0.05,NS

Table 4: Comparison of cardiac parameters in control and overweight subjects

In the above table, all the cardiac parameters except pulse pressure are statistically highly significant (p < 0.0001) in overweight subjects when compared with controls.

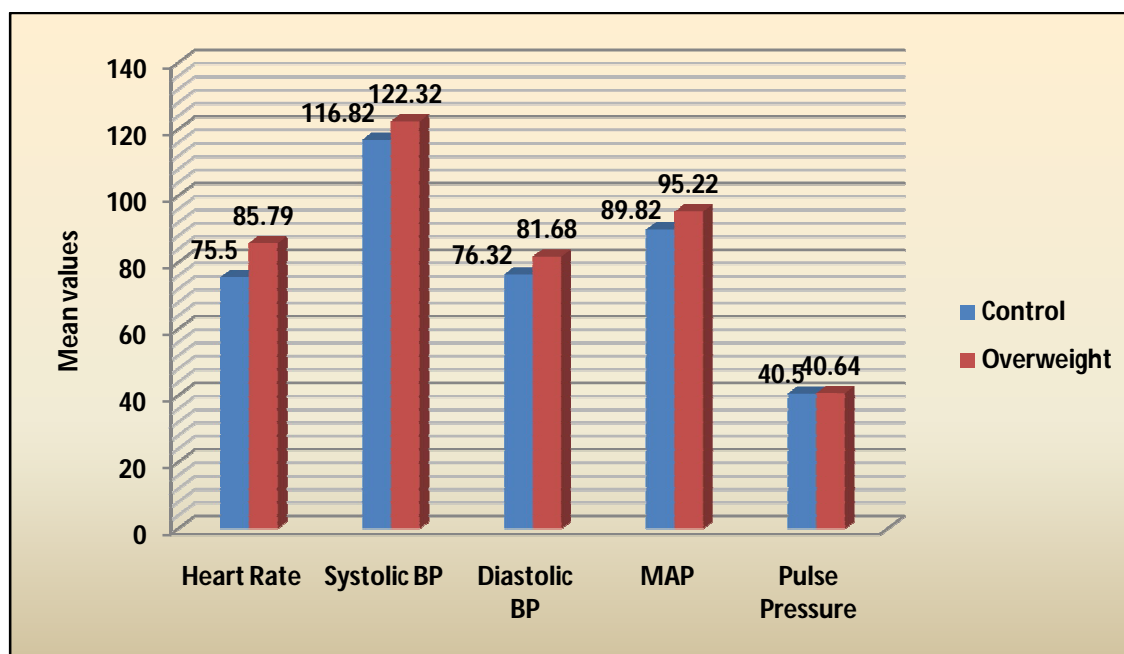


Figure 4: Cardiac parameters in control and overweight subjects.

Parameter	Control	Overweight	t-value	p-value
FVC Predicted (%)	94.86 ±3.83	92.51± 6.20	3.22	<0.01, S
PEFR (%)	97.21± 3.15	95.60± 4.88	2.76	<0.01, S

Table 5: Comparison of respiratory parameters in control and overweight subjects.

Above table shows pulmonary parameters FVC (t value=3.22, p value=0.0002) and PEFR (t value=2.76, p value=0.006) when compared between controls and overweight subjects are found statistically significant (p<0.01). values of pulmonary parameters are reduced in overweight subjects when compared with controls.

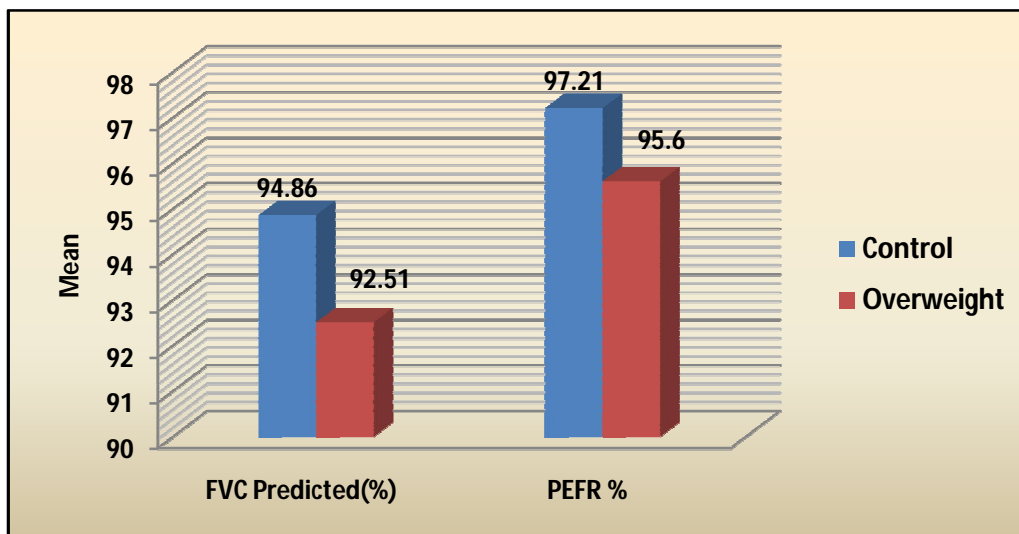


Figure 5: Respiratory parameters in control and overweight subjects.

Parameter	Female	Male	t-value	p-value
Heart rate	84.21± 5.19	85.14± 4.71	3.62	<0.01,S
SBP	121.94± 5.81	122.55 ±4.90	2.43	<0.01,S
DBP	81.63 ±3.94	81.70 ± 3.48	3.72	<0.01,S
MAP	95.07 ± 3.65	95.32 ± 2.98	2.63	<0.01,S
PP	40.31 ±4.93	40.83± 5.71	0.46	>0.05,NS

Table 6: Comparison of cardiac parameters in overweight female and male subjects

Above table shows change in all the cardiac parameters in overweight when compared in both, males and females by unpaired t test. It is found that there is a statistically significant increase in heart rate (t value= 3.62, p value= 0.002), systolic blood pressure (t value= 2.43, p value= 0.005), diastolic blood pressure (t value= 3.72, p value= 0.002), mean arterial pressure (t value= 2.63, p value= 0.005) in male when compared with female overweight subjects. But, difference in pulse pressure (t value=0.46, p value= 0.64) between males and females is found to be non-significant.

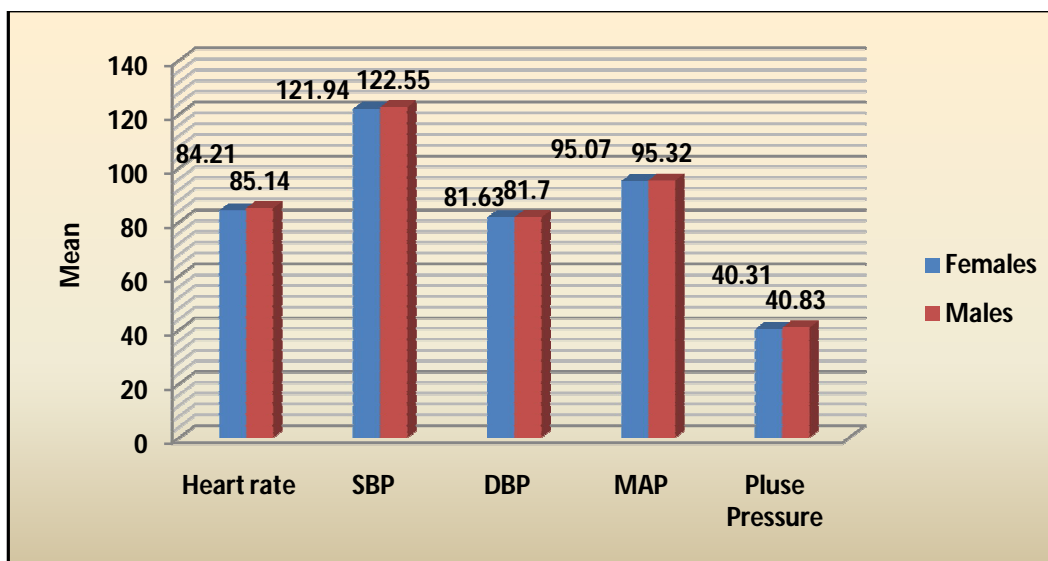


Figure 6: Cardiac parameters in overweight female and male subjects

Parameter	Male	Female	t-value	p-value
FVC Predicted (%)	94.18 ± 4.91	89.81± 7.14	3.62	<0.01, S
PEFR (%)	96.61± 4.58	93.95± 4.95	2.73	<0.01,S

Table 7: Comparison of Respiratory parameters in male and female of overweight subjects

Above table show changes in the respiratory parameters in overweight when compared in both, males and females by unpaired t test. It is found that there is more lower values of FVC (t value=3.62, p value= 0.002) and PEFR (t value= 2.73, p value= 0.005) in female when compared to male. It is found statistically significant.

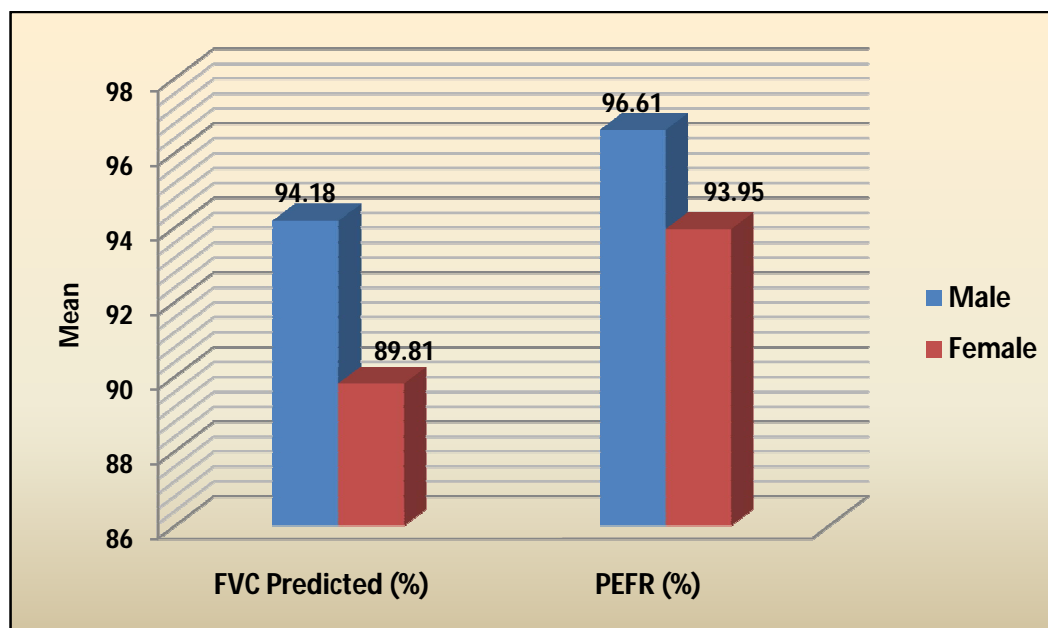


Figure 7: Respiratory parameters in male and female of overweight subjects

Parameter	Controls		Overweight	
	r-value	p-value	r-value	p-value
Heart rate	0.1329	0.187455	0.2789	<0.01,S
SBP	0.1506	0.134755	0.3002	<0.01,S
DBP	0.1914	0.056441	0.2131	<0.01,S
MAP	0.2363	0.017937	0.3081	<0.01,S
PP	-0.0162	0.872902	0.1296	>0.05,NS

Table 8: Correlation of BMI with Cardiac parameters in Controls and Overweight subjects

Above table shows that in overweight subjects, there is positive correlation between body mass index (BMI) with change in heart rate (r value= 0.2789, p value= 0.004955), systolic blood pressure (r value= 0.3002, p value= 0.002409), diastolic blood pressure (r value= 0.2131, p value= 0.033277) and mean arterial pressure(r value= 0.308, p value= 0.0018) which is found statistically significant.

Parameter	Controls		Overweight	
	r-value	p-value	r-value	p-value
FVC	0.092	0.36263	-0.021	>0.05,NS
PEFR	0.14	0.164756	-0.0753	>0.05,NS

Table 9: Correlation of BMI with respiratory parameters in Controls and Overweight subjects

In the above table, on comparing respiratory parameters in overweight subjects, it is found that there is negative correlation (statistically non-significant) between body mass index (BMI) and change in FVC(r value= -0.021, p value= 0.8357) and PEFR (r value= -0.0753, p value= 0.4565)which is found statistically non-significant.

5. Discussion

The present study was carried out to assess certain cardio-respiratory parameters in 100 healthy controls and 100 overweight subjects of 18-22 years of age group with special reference to anthropometric indices in first year medical students. Medical students were specifically chosen for this research, as creating awareness among them would be projected at a higher degree on society by the subjects themselves.

This age group 18-22 years was chosen because they are at the junction of adolescence and adulthood, and this phase of life could help us track the future progression of obesity related disorders. There is little evidence of data on research conducted in this age population.

After thorough physical examination of cases and controls, data was collected and statistics was applied.

Various anthropometric and cardio-respiratory parameters studied and data analyzed is discussed under the following headings. Data is expressed as Mean \pm SD and p value <0.05 is considered statistically significant.

The mean value \pm standard deviation (Mean \pm S.D.) of BMI is found to be 20.97 ± 1.95 in controls (Group A) and 25.94 ± 0.90 in overweight (Group B) (Table No.III, Graph No.III) When unpaired t- test is applied, it is found to be statistically highly significant (t value= 23.14, p value < 0.0001).

5.1. Cardiac Parameters

5.1.1. Heart Rate(beats/min)

The mean heart rate in controls (Group A) is 75.5 ± 5.04 . The mean heart rate (beats/min) in overweight subjects (Group B) is 84.79 ± 4.89 . There is statistically significant increase in the heart rate in overweight subjects compared to controls. (t value = 13.22, p value < 0.0001 HS). (Table No IV, Figure IV)

There is a positive correlation between BMI (body mass index) and heart rate. Increase in BMI increases heart rate. Similar findings were reported by multiple studies like Alberto Salvadori et al.^x, Krzysztof Narkiewicz et al.^{xi} and Gilles Paradis et al.^{xii}

Body fat and the activity of the autonomic nervous system a study by Hugh R. Peterson et al.^{xiii} and others in 56 obese men showed that heart rate is directly related to the percentage of body fat.

Satipati Chatterjee et al.^{xiv} assessed cardio-respiratory fitness of obese boys and found that the oxygen consumption per kg of body mass was significantly higher among non-obese boys. There was also a significant increase in heart rate in obese boys as compared to non-obese boys.

Overweight causes activation of sympathetic nervous system. The autonomic nervous system is an important contributor to the regulation of both the cardiovascular system and energy expenditure. Heart rate increases with increase in percentage of body fat. A 10% increase in body weight is associated with a decline in parasympathetic tone and it is accompanied by a rise in mean heart rate and vice versa. Therefore, weight reduction decreases heart rate, which in turn reduces mortality.^{xv}

Body fat and sympathetic nerve activity in healthy subjects were studied by Urs scherrer et al.^{xvi} in 37 healthy subjects and they found that the resting rate of sympathetic nerve discharge to skeletal muscle was directly correlated with BMI and percent of body fat. Overweight associated sympathetic activation could represent one potential mechanism contributing to the increased incidence of cardiovascular complications in overweight subjects.

5.1.2. Blood Pressure

a) Systolic Blood Pressure: There is a statistically significant increase (t value = 8.28, p value <0.0001) in systolic blood pressure in overweight subjects (122.32 ± 5.25 mm of Hg) when compared to controls. (116.82 ± 4.06 mm of Hg) (Table IV, Figure IV)

b) Diastolic Blood Pressure: In the present study, there is a statistically significant increase (t value = 9.97, p value <0.0001) in diastolic blood pressure in overweight subjects (81.68 ± 3.65 mm of Hg) when compared to controls (76.32 ± 3.95 mm of Hg) (Table IV, Figure IV)

i) Mean arterial Pressure: In our study, there is a statistically significant increase (t value = 12.19, p value <0.0001) in mean arterial pressure in overweight subjects (95.22 ± 3.23 mm of Hg) when compared to controls (89.82 ± 3.03 mm of Hg) (Table IV, Graph No.IV)

ii) Pulse Pressure: In the the present study, there is a slight increase in pulse pressure in overweight subjects (40.64 ± 5.66 mm of Hg) when compared to controls (40.50 ± 5.48 mm of Hg), which is found statistically non- significant. (t value = 0.17, p value = 0.86) (Table IV, Graph no.IV)

- Correlation of BMI with Cardiac Parameters:

In the the present study, on statistical analysis, it is found that there is a positive correlation between BMI (Body Mass Index) and change in heart rate (r value= 0.2789, p value= 0.004955), systolic blood pressure (r value=0.3002, p value=0.002409), diastolic blood pressure (r value= 0.2131, p value= 0.033277) and mean arterial pressure (r value= 0.3081, p value= 0.001818). However, we could not find a statistically significant correlation between BMI and pulse pressure. (Table XI)

As BMI increases, change in systolic and diastolic blood pressure also increases in overweight subjects.

Similar findings were reported by multiple studies, Rose Stamler et al.^{xvii}, Efrain Reisin et al.^{xviii}, Fronz H. Messerli^{xix}, William B Kannel et al.^{xx}, Roland T Jung^{xxi}, Mario Vaz et al.^{xxii}, Babu B. V. et al.^{xxiii}, Krzysztof Narkiewicz M et al.^{xxiv}, Alan R at al^{xxv}, Gilles Paradis et al.^{xii}.

Ravisankar P et al.^{xxvi} (2005) assessed correlation between BMI and blood pressure indices; handgrip strength and handgrip endurance among overweight adolescents and found that SBP, DBP and mean arterial pressure were lowest in underweight and highest in overweight subjects. Heart rate was increased in overweight subjects.

As BMI increases, there are significant changes seen in cardio-respiratory parameters. Likewise, as BMI increases, change in systolic and diastolic blood pressure also increases. Similar findings have been reported by Nicola I. Abate et al.^{xxvii} (2001) and Clarice D. Brown et al.^{xxviii} (2000)

Body mass index (BMI), Kg/m², has a high correlation with body fat. The adipose tissue plays a causative role in the genesis of hypertension.^{xxix}

BMI depends on several variables, including behaviour patterns, nutritional habits, social ideals and also changes due to aging.^{xxx}

However, BMI does not adequately describe the distribution of fat (which may be more predictive of morbidity), nor does it directly measure body composition and muscle mass. Despite these limitations, the National Institutes of Health's "Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults –The evidence report"^{xxxvi}, published in 1998, operationally defined 'overweight' as a BMI of 25 kg/m² to 29.9 kg/m² and 'obesity' as a BMI of at least 30 kg/m². This definition is consistent with recommendations from the World Health Organization and most other countries.

WHO has recommended the use of body mass index (BMI) as the simplest form of defining obesity.

Epidemiological studies with longitudinal follow up have demonstrated association between simple anthropometric measures like height, weight and BMI (body mass index) and waist circumference with cardiovascular risks.^{xxxvii,xxxviii} Studies have linked alterations in autonomic nervous activity with increased cardiovascular risk.^{xxxiv}

Increase in BMI leads to increased in blood pressure by various mechanisms:

1) Increase in mass of fat causes insulin resistance. This results in hyperinsulinemia.^{xxxv} Insulin predisposes to hypertension by the following mechanisms^{xxxv} -

- Stimulating renal sodium reabsorption
- Stimulation of sympathetic nervous activity
- Increased secretion of endothelin
- Impaired vasodilatation

2) In Overweight persons, leptin levels increase due to adipocyte accumulation. It stimulates arcuate POMC (pro-opiomelanocortin) expression and α -MSH (melanocyte stimulating hormone), which would then act elsewhere in the hypothalamus on MC4-R – expressing neurons (melanocortin receptors), causing increased food intake and increased sympathetic activity. This may be a crucial role linking obesity and hypertension.^{xxxix} The overweight subjects have elevated sympathetic activity that may contribute significantly to obesity related to hypertension.^{xxxix}

The various mechanisms and mediators for elevated adrenergic activity are:

- Elevated insulin levels with insulin resistance
- Elevated leptin levels
- Activation of renal afferents stimulated by increased intra-renal pressures, due to structural and functional alterations in the kidney.
- Angiotensin II release
- Potentiation of central chemoreceptors sensitivity
- Impaired baroreceptor sensitivity

3) The renin angiotensin aldosterone system seems to be activated in obesity, despite volume expansion and sodium retention. Reports suggests that a positive relationship between plasma angiotensin level, plasma rennin activity, and plasma angiotensin converting enzyme with BMI exists in humans. Moreover elevated serum aldosterone levels have been reported in overweight.^{xxx}

4) Adipocytes act as a source of inflammatory cytokines, such as tumour necrosis factor- α , interleukin-6, C-reactive protein and plasminogen activator. Thus obesity acts as a low grade inflammatory condition. The progressive pro-inflammatory state resulting from increased obesity promotes insulin resistance and also perpetuates atherogenesis. The endothelial modulators such as vasoactive endothelial growth factor, plasminogen activator inhibitor-I, angiotensinogen, rennin and angiotensin II are secreted by fat cells that contribute to vasomotor dysfunction and cause hypertension and endothelial injury.^{xxxvi}

5.2. Parameters of Pulmonary Function Tests

5.2.1. FVC (Forced Vital Capacity)

It is the maximum volume of air which can be breathed out as forcefully, rapidly and completely as possible following a maximum inspiration. It is almost equal to vital capacity in normal subjects. It is expressed in litres at BTPS. It is an indirect measure of flow and resistive properties of the lungs and lung volume also. In the the present study, we observed a statistically significant decrease (t value = 3.22, p value = 0.0002) in forced vital capacity in overweight subjects (92.51 \pm 6.20) when compared to controls (94.86 \pm 3.83). (Table No.V, Graph No.V)

5.2.2. PEFr (Peak Expiratory Flow Rate)

It is defined as maximum expiratory flow rate sustained for at least 10 milliseconds during the forced expiratory manoeuvre. It is expressed in Litre/second at BTPS. It is influenced to a greater extent by narrowing of large airways. It depends on expiratory efforts exerted during forceful expiration as well as status of airways and it is mainly influenced by efficiency of expiratory muscle, elastic recoil pressure of lung and airway size.

In our study, we observed that PEFr show statistically significant decreases (t value= 2.76, p value= 0.006) in peak expiratory flow rate in overweight subjects (95.60 \pm 4.88) when compared to controls (97.21 \pm 3.15). (Table No.V, Graph No.V).

5.3. Correlation of BMI with Respiratory Parameters

In the present study, on statistical analysis, it is found that there is a negative correlation between BMI (Body Mass Index) and change in forced vital capacity (r= - 0.021, p=0.835711) and peak expiratory flow rate (r= - 0.0753, p= 0.456519) (Table No.XII)

Similar findings were reported by multiple studies, P S Thomas et al.^{xxxvii}, Rubinstein et al.^{xxxviii}, Judith et al.^{xxxix}, Y Chen and his associates^{xl}, Francoise Zerah et al.^{xli}, Hamid Sahebajami et al.^{xlii} and R A Watson et al.^{xliii}

Overweight influences upper airway reflexes, lung mechanics. It adversely affects chest wall mechanics, reduces FRC and ERV and causes a decrease in total respiratory compliance due to deposition of subcutaneous adipose tissue. These often result in a ventilation-perfusion (V/Q) mismatch, especially in the supine position. There is also a decrease in lung compliance due to increased pulmonary blood volume. 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 intra-abdominal adipose tissue, which interferes with the mechanical properties of the chest wall causing decrease in compliance and preventing full excursion of the diaphragm^{xliv}. There are also effects of obesity on upper airway tone and hence resistance, which add a mechanical load that increases the work of breathing. Morbid obesity may also induce restrictive disturbance of respiratory function, related to reduced compliance of the chest wall and or pulmonary parenchyma. Obese individuals have an increased demand for ventilation and breathing work load, respiratory muscle inefficiency, decreased functional reserve capacity and expiratory reserve volume, and closure of peripheral lung units.^{xlv}

Our study showed a negative correlation between BMI and lung functions similar to other studies, Banerjee J et al.^{xlv}, Morsi M G et al.^{xlvi}, Chen Y et al.^{xlvii}

The most common abnormalities in obese adults are decreased expiratory flow rates and lung volumes.

Spathapoulos et al.^{xlviii} reported that PFT parameters (FEV1, FVC, FEF25–75 and FEV1/FVC) were decreased in overweight and obese cases (high BMI). Also, the most common abnormality found was combined restrictive and obstructive type. This may be explained by extrinsic mechanical compression due to fat accumulation which causes decreased chest wall recoil and compliance resulting in expiratory volume reduction. Nevertheless, a mechanical effect was not the only factor since, pulmonary function parameters were affected by increased serum leptin levels in obese subjects.

5.4. Summary

The present study was conducted in the department of Physiology. Subjects were grouped into 100 overweight subjects having body mass index 25-29.9 kg/m² and 100 healthy controls having body mass index 18.50-24.99 kg/m². These include both males and females in age group of 18-22 years.

Cardio-respiratory parameters such as heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, pulse pressure and forced vital capacity and peak expiratory flow rate were assessed. Subjects suffering from any acute or chronic diseases or any other systemic illness that directly or indirectly affects cardio-respiratory parameters were excluded.

Cardiac parameters and pulmonary function tests were carried out on a mercury sphygmomanometer and on a computerized spirometer respectively with special reference to anthropometric indices. The following parameters were studied:

- Heart Rate
- Systolic Blood Pressure
- Diastolic Blood Pressure
- Mean arterial Pressure
- Pulse Pressure
- FVC
- PEFR

Results of above parameters were compared between 100 overweight subjects and 100 age and sex matched healthy normal controls. Results showed all the cardiac parameters, i.e. heart rate, systolic blood pressure and diastolic blood pressure were significantly increased in overweight subjects when compared with controls. While, respiratory parameters, i.e. FVC (forced vital capacity) and PEFR (peak expiratory flow rate) were reduced in overweight subjects when compared with controls.

6. Conclusions

The conclusions of our study are:

- An increased in body weight is associated with an increase in heart rate, the higher the body weight, the higher is the heart rate.
- An increase in body weight is associated with an increase in systolic blood pressure and diastolic blood pressure, the higher the body weight, the higher is the increase.
- An increase in body weight is associated with a decreased in forced vital capacity and the peak expiratory flow rate, the higher the body weight, the lower are these lung function parameters.
- There is a positive correlation between increase in body mass index (BMI) and change in heart rate, systolic blood pressure and diastolic blood pressure.
- There is a negative correlation between increase in body mass index (BMI) and change in forced vital capacity (FVC) and peak expiratory flow rate (PEFR).

The positive correlation between body mass index and change in systolic and diastolic blood pressures in overweight subjects is due to sympathetic overactivity. Overweight-related sympathetic overactivity is thought to be a compensatory mechanism to burn fat and minimize weight gain.

7. References

- i. Brown WJ, Mishra G, Kenardy J, Dobson A. Relationships between body mass index and well-being in young Australian women. *Int J Obes Relat Metab Disord*. 2000; 24(10):1360–68
- ii. Eckel RH, York DA, Rossner S, Hubbard V, Caterson I, St Jeor ST, et al..American Heart Association Prevention Conference VII : Obesity, a world- wide epidemic related to heart disease and stroke: executive summary. *Circulation* 2004; 10 :2968-75
- iii. Park K. Textbook of Preventive and Social Medicine.22nd Edition. Chapter 6. Epidemiology of non-communicable diseases, Obesity. Banarasidas Bhanot Publishers;2013:367-71
- iv. Unnikrishnan A, Kalra S, Garg MK. Preventing obesity in India: Weighing the options. *Indian J Endocrinal Metab*.2012 Jan-Feb;16(1):4-6.
- v. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004;363:157-63.
- vi. Clinical Guidelines on the identification, evaluation and treatment of Overweight and Obesity in adults: The evidence report: National Institutes of Health. *Obes Res*.1998; suppl 2:51 S-209S.
- vii. Pollack, Andrew. "A.M.A. Recognizes Obesity as a Disease". *New York Times*. Archived from the original (www.nytimes.com)on June 18, 2013.
- viii. Bray G A and Gray D S Obesity Part-I Pathogenesis. *West J. Med* 1988 Oct ; 149: 429-41
- ix. Taussig LM, Chernick V, Wood R. standardization of lung function testing in children : Proceeding and recommendation of the GAP conference committee. *J Pediatr* 1980;97:668-76.
- x. Salvadori A, Fanari P, Mazza P, Agosti R, Longhini E. Work Capacity and Cardiopulmonary adaptation of the obese subject during exercise testing. *Chest* 1992; 101:674-79
- xi. Narkiewicz K, Vande Borne PJH, Cooley RL, Dyken ME, Somers VK. Sympathetic activity in obese subjects with and without obstructive sleep apnea. *Circulation* 1998;98:772-76
- xii. Paradis G, Lambert M, O'Loughlin J, Lavallee C, Aubin J, Delvin E, et al.. Blood pressure and adiposity in children and adolescents. *Circulation*. 2004;110 (13):1832–38.
- xiii. Peterson HR, Rothschild M, Weinberg CR, Fell RD, Mcleish KR, Pfeifer MA.Body fat and the activity of the autonomic nervous system. *N Engl J Med* 1988;318: 1077-83
- xiv. Chatterjee S, Chatterjee P, Bandyopathayay A. Cardiorespiratory Fitness of obese boys. *Indian J Physiol Pharmacol* 2005;49(3):353-57.
- xv. La Rovere MT, Bigger JT, Marcus FI, Mortara A, Schwartz PJ. Baroreflex sensitivity and heart rate variability in prediction of total cardiac mortality after myocardial infarction. ATRAMI (Autonomic Tone and Reflexes After Myocardial Infarction) Investigators.*Lancet* 1998;351 : 478-84
- xvi. Scherrer U, Randin D, Tappy L, Vollenweider P, Jequier E, Nicod P. Body fat and sympathetic Nerve activity in healthy subjects. *Circulation* 1994;89:2634-50
- xvii. Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Weight and blood pressure: findings in hypertension screening of 1 million Americans.*JAMA* 1978; 240 : 1607-10
- xviii. Reisin E, Abel R, Modan M, Silverberg DS, Eliahou HE, Modan B. Effect of weight loss without salt restrictions on the reduction of blood pressure in overweight hypertensive patients. *N Eng J Med* 1978;298: 1-6
- xix. Messerili FH. Cardiovascular effects of obesity and Hpertension. *The Lancet* 1982;1165-68
- xx. Kannel WB, Agostino RBD, Cobb JL.Effect of weight on cardiovascular disease. *Am J Clin Nutr* 1996;63(S);419S-22S
- xxi. Jung R. Obesity as a disease. *British Medical Bulletin*. 1997;53 (2) : 307-21
- xxii. Babu BV, Kusuma YS, Naidu JM. The influence of age,sex and obesity on blood pressure levels in a tribal population. *Indian J Physiol Pharmacol* 1998;42 (4):543-47
- xxiii. Babu BV, Kusuma YS, Naidu JM. The influence of age,sex and obesity on blood pressure levels in a tribal population. *Indian J Physiol Pharmacol* 1998;42 (4):543-47
- xxiv. Narkiewicz K, Vande Borne PJH, Cooley RL, Dyken ME, Somers VK. Sympathetic activity in obese subjects with and without obstructive sleep apnea. *Circulation* 1998;98:772-76
- xxv. Sinaiko AR, Donahue RP, Jacobs DR, Prineas RJ. Relation of weight and rate of increase in weight during childhood and adolescence to body size, blood pressure, fasting insulin and lipids in young adults. *Circulation* 1999;99:1471-76
- xxvi. Ravisankar P, Madanmohan, Udupa K, Prakash ES. Correlation between body mass index and blood pressure indices, handgrip strength and handgrip endurance in underweight, normal weight and overweight adolescents. *Indian J Physiol Pharmacol*. 2005;49(4):455-61
- xxvii. Abate NI, Mansour YH, Tuncel M, Arbiqye D, Chavoshan B, Kizilbash A, et al.. Overweight and Sympathetic Overactivity in Black Americans. *Hypertension*.2001;38:379-83
- xxviii. Brown C D, Higgins M, Donato K A, Rohde F C, Garrison R, Obarzanek E, et al.. Body mass index and the Prevalence of Hypertension and Dyslipidemia. *Obesity Research* 2000;8:605-19
- xxix. Aneja A, Fadi EL-Atat, Samy I, MC Farlane and Sowers J R. Hypertension and obesity. *Recent Progress in Hormone Research* 2004;59:169-205
- xxx. Paknahad Z, Omidvar N, Mahboub S, Afiatmilani S, Ostadrahimi AR, Ebrahimi M. Body mass index of reproductive age group women and its relationship with iron status. *Journal of Tabriz University of Medical Sciences*. 2001;35(51):17–23

- xxxvi. National Institutes of Health Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults – The evidence report. National Institutes of Health. *Obes Res.* 1998;6(Suppl 2):51S–209S.
- xxxvii. Pischon T, Boeing H, Hoffmann K, et al.. General and abdominal adiposity and risk of death in Europe. *The New England Journal of Medicine.* 2008;359(20):2105–20.
- xxxviii. Gonzalez AB, Hartge P, Cerhan JR, et al.. Body-Mass Index and mortality among 1.46 million white adults. *New England Journal of Medicine.* 2010;363:2211–19
- xxxix. Curtis BM, O'Keefe JH. Autonomic tone as a cardiovascular risk factor: the dangers of chronic fight or flight. *Mayo Clinic Proceedings.* 2002;77(1):45–54.
- xl. Vikrant S, Tiwari S C. Essential Hypertension- Pathogenesis and Pathophysiology. *Journal Indian Academy of Clinical Medicine.* July-Sept 2001;2(3):140-61
- xli. Redinger R N. The Pathophysiology of obesity and its Clinical Manifestations. *Gastroenterology and Hepatology Nov* 2007;3(11):856-63
- xlii. Thomas PS, Owen ERTC, Hulands G, Milledge JS. Respiratory function in the morbidly obese before and after weight loss. *Thorax* 1989;44:383-86
- xliiii. Rubinstein, Zamel N, Dubarry L, Hoffstein V. Airflow limitation in morbidly obese, nonsmoking men. *Annals of internal Medicine* 1990;112: 828-32
- xliiiii. Leech J, Onal E, Arosen R, Lopata M. Voluntary hyperventilation in obesity hypoventilation. *Chest* 1991;100: 1334-38
- xl. Chen Y, Horne SL, Dosman JA. Body weight and weight gain related to pulmonary function decline in adults : a six year follow up study. *Thorax* 1993;48:375-80
- xli. Zerah F, Harf A, Perlemuter L, Corino H, Lorino Am, Atlan G. Effect of obesity on respiratory resistance. *Chest* 1993;103:1470-76
- xlii. Sahebajami H, Gartside PS. Pulmonary function in obese subjects with a normal FEV1/FVC ratio. *Chest* 1996; 110 : 1425-29
- xliiii. Watson RA, Pride NB. Postural changes in lung volumes and respiratory resistance in subjects with obesity. *J Appl Physiol* 2005;98:512-17
- xliv. Collins LC, Hoberty PD, Walker JF, Fletcher EC, Peiris AN. The Effect of Body Fat Distribution on Pulmonary Function tests. *Chest* 1995;107:1298-02
- xl. Banerjee J, Roy A, Singhamahapatra A, Dey PK, Ghosal A, Das A. Association of body mass index with lung function parameters in non-asthmatics identified by spirometric protocols. *J Clin Diagn Res* 2014;8:12-4.
- xlvi. Morsi MG. Abdominal and total body adiposity markers in asthmatic patients. *J Med Sci* 2009;9:59-69
- xlvii. Chen Y, Horne SL, Dosman JA. Body weight and weight gain related to pulmonary function decline in adults : a six year follow up study. *Thorax* 1993;48:375-80
- xlviii. Spathopoulos, E. Paraskakis, G. Trypsianis, The effect of obesity on pulmonary lung function of school aged children in Greece, *Pediatr. Pulmonol.* 44 (2009) 273–80