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Original article

Correlation between body mass index and cardiovascular parameters in obese and non-obese in different age groups

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A R T I C L E I N F O

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A B S T R A C T

Aims: In obesity, as excessive adipose tissue accumulates, an altered metabolic profile occurs along with a variety of adaptations and alterations in cardiovascular structure and function even in the absence of co-morbidities. This study was undertaken to analyze the differences in certain well-defined cardiovascular parameters in obese and non-obese subjects in the age groups of 21-60 years. Methods: 117 healthy male obese subjects with Body Mass Index 30 kg/m2 and also 110 healthy male non-obese with Body Mass Index of 18.50 – 24.99 kg/m2 were selected. Parameters such as heart rate, systolic blood pressure, diastolic blood pressure, mean blood pressure and pulse blood pressure were assessed. Results: In our study, there was a statistically significant increase in heart rate, systolic blood pressure and diastolic blood pressure in obese subjects when compared to non-obese in all age group. There was a positive correlation between body mass index and heart rate, systolic blood pressure, diastolic blood pressure, mean blood pressure and pulse blood pressure. The degree of rise was higher for the systolic blood pressure than the diastolic blood pressure. Conclusions: Although our study is by no means exhaustive, it does provides a glimpse into the variety of adaptations in cardiovascular structure and function that occur as excessive adipose tissue accumulates, even in the absence of overt disease.

1. Introduction

The price we are paying for an affluent and developed society is a sedentary life style and faulty dietary habits which result in an imbalance between energy intake and energy expenditure, which, in turn leads to obesity. Over weight and obesity represent a rapidly growing threat to the healthy populations in an increasing number of countries [1].Obesity is becoming a global epidemic, [2] and in the past 10 years in Europe and the United States, dramatic increase in obesity have occurred in both children and adults[3]. Through the use of Body Mass Index (BMI), the epidemic of obesity that began in the 1980s has been tracked through the end of the century. The original alarm was sounded in 1994 by the National Center For Health Statistics in USA when they reported data from the National Health And Nutrition Examination Survey (NHANES) [4]. The authors observed that over a span of 10 years from 1988 to 1999, the prevalence of overweight in adults increased from 55.95 to 64.5%.During the same period, the prevalence of obesity increased from 22.9% to 30.5% [5].

Obesity is associated with an increased risk of morbidity and mortality as well as reduced life expectancy. Complications are either directly caused by obesity or indirectly related through mechanisms sharing a common cause such as a poor diet or a sedentary lifestyle. Overweight and obesity may account for as many as 15-30% of deaths from Coronary Heart Disease (CHD) and 65-75% of new cases of type 2 Diabetes Mellitus [6]. The strength of the link between obesity and specific conditions varies. One of the strongest is the link with type 2 diabetes. Excess body fat underlies 64% of cases of diabetes in men and 77% of cases in women [6].

Concern grows that the current dramatic rise of obesity among adolescents portends a future wave of increasing cardiovascular disease as these overweight youth reach the adult years. The direct

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effects of the obese state on heart function, and the means by which excessive body fat might negatively affect cardiac health during the growing years, however, has received less attention, so priority is given in this study to assess cardiac parameters in young adults also. Over weight and obesity are associated with an increased risk of disabling conditions such as arthritis and impaired quality of life in general. A variety of adaptations in cardiorespiratory structure and function occur in the individual as adipose tissue accumulates in excess amounts, even in the absence of co-morbidities [7]. 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 apnea, hypoventilation, and the prothrombotic state, in addition to as yet unrecognized mechanisms. The cardiovascular disorders due to obesity result in increased mortality from complications such as coronary artery disease, heart failure, arrhythmias and sudden death [8]. As a result, they were given more priority in this study, wherein some of the cardiac parameters were assessed in an attempt to highlight the complications because obesity is one of the leading preventable causes of death worldwide.

2. Materials and Methods

117 male obese subjects and also 110 male non-obese controls were selected from the general population of Davangere randomly. Healthy males with BMI 30 kg/m2 in the age group of 21-60 years were classified as obese. Healthy males with BMI of 18.50 – 24.99 kg/m2 in the age group of 21-60 years were included as controls in this study. Subjects with BMI between 25-29.9, those less than 20 years of age or above 60 years of age and female individuals were excluded from this study. Subjects with medical problems especially cardiorespiratory illnesses and smokers were also excluded from the study. All the subjects gave an informed consent after detailed procedure of the non-invasive technique was explained to them. A brief history including smoking history and a clinical examination of the cardiovascular system and respiratory system was done to exclude medical problems and to prevent confounding of results. Physical examination of all subjects included measuring height in meters, weight in kilograms and Body mass index was derived by Quetelet's index - weight (kg)/ height (m2). Resting heart rate was recorded by using 12 lead ECG and blood pressure measurement with a mercury sphygmomanometer using the appropriate sized cuff. Cardiac parameters like and heart rate, systolic blood pressure, diastolic blood pressure, mean blood pressure and pulse blood pressure were assessed.

2.1. Statistical Analysis

Descriptive data are presented as Mean Standard Deviation and Range values. Unpaired student's t-test was used for two groups comparison. Pearson's correlation coefficient was used to measure the relationship between the measurements.

A p-value of 0.05 or less was considered for statistical significance.

3.Results

Out of the hundred and ten non-obese subjects, 20 subjects were in the age group of 21-30 years, 31 subjects were in the age group of 31-40 years, 24 subjects were in the age group of 41-50 years, 35 subjects were in the age group of 51-60 years. Out of the hundred and seventeen obese subjects, 21 subjects were in the age group of 21-30 years, 30 subjects were in the age group of 31-40 years, 27 subjects were in the age group of 41-50 years, 39 subjects were in the age group of 51-60 years.

On analysis of the physical characteristics of the 110 non-obese subjects, the mean age (years) was 39.6 ± 6.7; the mean weight (kg) was 62.3 ± 9.8; the mean height (mt) was 1.67± 0.1; the mean BMI (kg/m2) was 22.4 ± 1.8 and the mean BSA (m2) was1.71 0.2.On analysis of the physical characteristics of the 117 obese subjects, the mean age (years) was 38.8± 5.2; the mean weight (kg) was 81.1 ± 7.8; the mean height (mt) was 1.59± 0.1; the mean BMI (kg/m2) was 32.1 ± 1.9 and the mean BSA (m2) was 1.83± 0.1.

Comparison of cardiac parameters like heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean blood pressure (MAP) and pulse blood pressure (PP) between non-obese and obese showed a statistically highly significant change between obese and non obese. [Table 1] Comparison of age - related changes in heart rate (beats / min) between non-obese and obese showed a statistically highly significant increase in obese compared to non-obese. [Fig.1] Comparison of age - related changes systolic blood pressure (mm of Hg) between non-obese and obese showed a statistically highly significant increase in obese compared to non-obese. [Table 2] The degree of rise in SBP was more than the rise in DBP. There was statistically significant increase in SBP in obese subjects compared to non-obese in all age groups. (P value < 0.001) Comparison of age - related changes in diastolic blood pressure (mm of Hg) between non-obese and obese. There was statistically significant increase in DBP in obese subjects compared to non-obese subject with age group of 21-30, 41-50 year and 51-60 years (P value < 0.001). [Table 3] Even though there was a increase DBP between obese and non-obese in the age groups 31-40 years, it was not statistically significant. Correlation between various cardiac parameters and BMI in obese, showed that there was a statistically significant positive correlation between various cardiac parameters and BMI. [Table 4]

4.Discussion

Obesity is often defined simply as a condition of abnormal or excessive fat accumulation in adipose tissue, to the extent that health may be impaired. Body Mass Index provides the most useful, albeit crude, population-level measure of obesity. It can be used to estimate the prevalence of obesity within a population and the risks associated with it. Obesity produces an increment in total blood volume and cardiac output that is caused in part by the increased metabolic demand induced by excess body weight [10]. The increase in blood volume in turn increases venous return to the heart, increasing filling pressures in the ventricles and increasing wall tension. This leads to left ventricular hypertrophy and this can decrease the diastolic compliance of the ventricle which can further progress to diastolic dysfunction and as wall tension increases further, can lead to systolic dysfunction. Thus through different mechanisms like increased total blood volume, increased cardiac output, left ventricular hypertrophy and further diastolic dysfunction, obesity may predispose to heart failure [10].

Table –1: Comparison of cardiac parameters between non-obese and obese

Groups	HR beats/min		SBP (mm of Hg)		DBP (mm of Hg)		MAP (mm of Hg)		PP (mm of Hg)	
Groups	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD
Non-obese BMI 18.50-24.99	68-87	81.4 ± 4.6	112-138	126.8 ± 5.8	60-90	74.1 ± 5.1	77-104	91.7± 4.6	34-64	52.6 ± 5.7
Obese BMI ≥ 30	80-96	90.1± 3.5	124-154	142.4 ± 7.9	66-94	80.7 ± 8.3	86-114	104.3 ± 8.0	50-66	56.8 ± 4.0
Mean diff	8.7		15.6		6.6		12.6		4.2	
	t	15.1	15.9		7.8		13.7		5.93	
Significance	P	< 0.001 HS	< 0.001 HS		< 0.001 HS		< 0.001 HS		< 0.001 HS	

All values expressed as Mean SD
Analysis for all parameters done by Unpaired't' test
HS – Highly Significant, S – Significant and NS – Not Significant.

Table - 2: Comparison of age - related changes in systolic blood pressure (mm of Hg) between non-obese and obese.

Age group (yrs)	Non-Obese		Obese		Significance	
	No. of cases	Mean ± SD	No. of cases	Mean ± SD	t	p
21 –30	20	118.6±6.2	21	124.9± 1.2	4.22	< 0.001 HS
31 – 40	31	116.7± 5.2	30	124.9± 1.1	4.99	< 0.001 HS
41 – 50	24	127.6± 4.8	27	140.4± 6.1	10.03	< 0.001 HS
51 – 60	35	129.1± 3.7	39	148.4± 3.6	25.2	< 0.001 HS

All values expressed as Mean SD.
Analysis for all parameters done by Unpaired't' test
HS – Highly significant, S – Significant, NS – Not Significant

Table –3: Comparison of age - related changes in diastolic blood pressure (mm of Hg) between non-obese and obese.

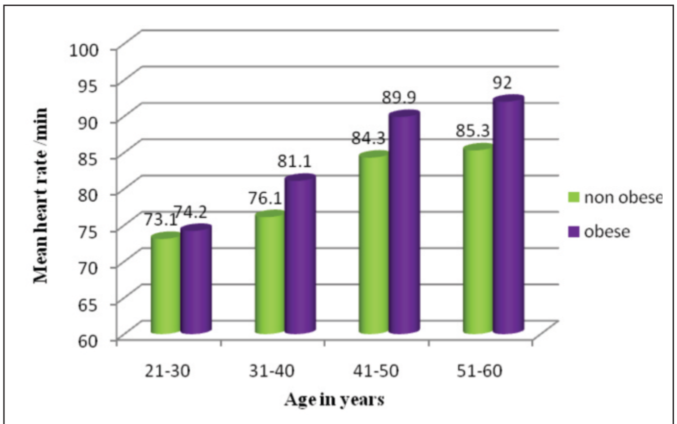
Age group (yrs)	Non-Obese		Obese		Significance	
	No. of cases	Mean ± SD	No. of cases	Mean ± SD	t	p
21 – 30	20	66.9 ±1.3	21	67.8±4.1	4.3	< 0.001 HS
31 – 40	31	65.1± 4.3	30	67.1± 1.1	1.50	0.16 NS
41 – 50	24	73.3± 1.3	27	83.3 ± 6.7	10.5	< 0.001 HS
51 – 60	35	75.9± 4.4	39	92.2± 1.3	28.1	< 0.001 HS

All values expressed as Mean SD.
Analysis for all parameters done by Unpaired 't' test
HS – Highly significant, S – Significant, NS – Not Significant

Table 4: Correlation of Body Mass Index with cardiac parameters

Correlation between	Obese subjects		Normal subjects	
Groups	Pearson's r -value	p-value	Pearson's r -value	p-value
BMI HR	+ 0.90	0.001 HS	+ 0.89	0.001 HS
BMI SBP	+ 0.67	< 0.01 HS	- 0.20	< 0.05 S
BMI DBP	+ 0.24	0.05 S	0.03	0.77 NS
BMI MAP	+ 0.20	< 0.05 S	- 0.06	0.55 NS
BMI PP	+ 0.25	< 0.05 S	- 0.23	< 0.05 S

Fig.1: Comparison of age - related changes in heart rate (beats/min) between non-obese and obese



In our study, there was a statistically significant increase in heart rate in obese subjects when compared to non-obese when each age subgroup was compared. There was a positive correlation with increasing body mass index causing increasing heart rate. Activation of the sympathetic nervous system occurs early in the course of obesity and the autonomic nervous system is an important contributor to the regulation of both the cardiovascular system and energy expenditure [11]. Hirsch and his colleagues studies also showed that heart rate increases with increase in percentage of body fat. A 10% increase in body weight is associated with a decline in parasympathetic tone accompanied by a rise in mean heart rate and conversely, heart rate declines during weight reduction [12].

Body fat and the activity of the autonomic nervous system was studied by Hugh R. Peterson and others in 56 healthy obese men, and showed that heart rate was directly related to the percentage of body fat [13].

Body fat and sympathetic nerve activity in healthy subjects were studied by Urs scherrer et al 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 [14].

In our study, there was a statistically significant increase in systolic blood pressure in obese subjects when compared to non-obese subjects with a mean increase of SBP by 15.4 mm Hg. Similarly, there was a statistically significant increase in diastolic BP in obese subjects when compared to non-obese with a mean increase of DBP by 6.6 mm Hg. It must be noted that in obese, DBP did not rise as much as the SBP did. There was a similar change observed when each age subgroup category was compared. There was also a positive correlation with increasing BMI causing further consistent increases in SBP, DBP and MAP. Factors linking obesity to increase in BP includes the increment in total blood volume and cardiac output that is caused in part by the increased metabolic demand induced by excess body weight and also mechanisms linking obesity and an increase in peripheral vascular resistance: endothelial dysfunction, insulin resistance, increased sympathetic nervous system activity, substances released from adipocytes (IL-6, TNF- and so forth) and sleep apnea [15]. Characteristically, obese subjects have increased sympathetic nerve activity, increased insulin levels and increased activity of the renin–angiotensin-aldosterone system [16].

Regional sympathetic nervous system activity and oxygen consumption in obese normotensive human subjects was studied by Mariovaz et al and found that diastolic blood pressure was significantly higher in the obese compared with lean subjects [17]. Babu B.V. and his colleagues studied the influence of obesity on blood pressure levels, indicated that the systolic blood pressure increases significantly along with BMI and triceps skin fold thickness. Although diastolic blood pressure has a positive correlation with BMI, it is not statistically significant [18].

Blood pressure and adiposity in children and adolescents were assessed by Gilles Paradis et al and showed that heart rate was increased, which suggests some degree of increased sympathetic activity. BMI was consistently associated with increase in SBP and DBP in all age-gender groups [19]. Obesity is commonly associated with hypertension, increased blood volume and cardiac output. There is also activation of the adrenergic system [20].

Correlation between BMI and blood pressure indices; handgrip strength and handgrip endurance among overweight adolescents was assessed by Ravi Sankar P and his coworkers and found that SBP, DBP and mean arterial pressure were lowest in under weight and highest in overweight subjects. Heart rate was increased in overweight subjects [21].

5.Conclusions

Though our study is by no means exhaustive, it does provides a glimpse into the variety of adaptations/alterations in cardiovascular structure and function that occur as excessive adipose tissue accumulates, even in the absence of overt disease. Although we understand to some extent the pathophysiology in overweight/obesity, a number of scientific questions need to be addressed for us to have a more complete understanding of this condition. Further research is recommended to understand how genes and gene-environment interaction leads to obesity. A better understanding of ethnic/racial differences in the development and progression of obesity is needed. We need to evaluate the strategies, and efficacy of obesity treatment. Policy research is needed on the impact of overweight/obesity on future health care in people.

6.References

[1] Park K. Park's textbook of preventive and social medicine. 18th ed. Jabalpur: M/S Bandrsidas Bhanot . 2005;316-319.

[2] World Health Organization. Obesity : Preventing and managing the global epidemic. WHO technical report series No. 894. Geneva. 2000.

[3] Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, Koplan JP. The spread of the obesity epidemic in the United States, 1991-1998. JAMA. 1999;282:1519-1522.

[4] Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults : the National Health and Nutrition Examination Surveys, 1960 to 1991. JAMA. 1994; 272:205-211.

[5] Flegal KM, Carrol MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. JAMA. 2002; 288:1723-1727.

[6] Jousilahti P, Tuomilehto J, Vartiainen E, Pekkanen J, Puska P. Body weight, cardiovascular risk factors, and coronary mortality: 15 year follow-up of middle-aged men and women in castern Finland. Circulation. 1996; 93:1372-79.

[7] Kasper DL, Braundwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL. Harrison's Principles of Internal Medicine vol.1, 16th ed. United States of America: The McGraw Hill Companies. 2005:422-429.

[8] 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 2:51S-209S.

[9] Alpert MA. Obesity cardiomyopathy; pathophysiology and evolution of the clinical syndrome. Am J Med Sci. 2001; 321:225-236.

[10] Kaltman AJ, Goldring RM. Role of circulatory congestion in the cardiorespiratory failure of obesity. Am J Med. 1976; 60:645-653.

[11] Benjamin EJ, Levy D. Why is left ventricular hypertrophy so predictive of morbidity and mortality? Am J Med Sci. 1999; 317:168-1 75.

[12] Hirsch J, Leibel RL, Mackintosh R, Aguirre A. Heart rate variability as a measure of autonomic function during weight change in humans. Am J Physiol. 1991; 261:1418-23.

[13] 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; 318:1077-1083.

[14] Scherrer U, Randin D, Tappy L, Vollenweider P, Jequier E, Nicod P. Body fat and sympathetic nerve activity in healthy subjects. Circulation. 1994; 89:2634-2640.

[15] Poirier P, Lemieux I, Mauriege P, Dewailly E, Blanchet C, Bergeron J, Despres JP. Impact of waist circumference on the relationship between blood pressure and insulin: the Quebec Health Survey. Hypertension. 2005; 45:363-367.

[16] Tuck ML, Sowers J, Dornfeld L, Kledzik G, Maxwell M. The effect of weight reduction on blood pressure, plasma rennin activity, and plasma aldosterone levels in obese patients. N Engl J Med. 1981; 304:930-933.

[17] Vaz M, Jennings G, Turner A, Cox H, Lambert G, Esler M. Regional sympathetic nervous activity and oxygen consumption in obese normotensive human subjects.Circulation. 1997; 96:3423-29.

[18] 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)543-547.

[19] Paradis G, Lambert M, O'Loughlin J, Lavallee C, Aubin J, Delvin E. Blood pressure and adiposity in children and adolescents. Circulation. 2004;110:1832-38.

[20] Opie LH.Heart physiology, from cell to circulation. 4th ed. Lippincott: Williams and Wilkins. 2004;453.

[21] Ravisankar P, Mohan M, Udupa K, Sankarnarayana EP.Correlation between BMI and blood pressure indices, handgrip strength and handgrip endurance in under weight, normal weight and over weight adolescents. Indian J Physiol Pharmacol. 2005;49(4):455-461.