Harsukh Educational Charitable Society
International Journal of Community Health and Medical Research
Journal home page: www.ijchmr.com doi: 10.21276/ijchmr

ISSN E: 2457-0117 **ISSN P:** 2581-5040 **Index Copernicus** ICV 2018=62.61

ORIGINAL ARTICLE

A Hospital Based Prospective Study to Evaluate the Effect of Obesity on Cardiovascular Autonomic Functions in School Children

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ABSTRACT:

Background: Obesity occurs when caloric intake exceeds energy expenditure and the excess calories are stored in an adipose tissue. Several studies in literature suggest that autonomic nervous system of obese individuals is chronically altered. So this study is done to investigate the cardiovascular autonomic nervous functions in obese school children. **Material & Methods:** 150 School children were randomly selected to obtain mixed group of children belonging to mixed socioeconomic status of age group 9-16 from various schools of Bikaner. Then they were screened to exclude underweight children by using lower cut-off limits of BMI. 50 underweight children were excluded and remaining 100 children were selected for the study. Now these 100 children were again segregated into two groups: Obese group (22) and Non obese group (78). The subject was asked to relax in supine position for 30 minutes. The resting heart rate was recorded on a standard ECG from lead II, at a paper speed of 25 mm/sec. The cardiovascular tests performed. **Results:** The comparison of mean value of parasympathetic and sympathetic activity in boys and girls shows in table no. 2. **Conclusion:** We concluded that parents of such obese children should take this very seriously and must guide their kids to go for physical exercise as well as diet control to check the rapidly increasing weight.

KeyWords: Obesity, Children, BMI, Cardiovascular activity

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This article may be cited as: Maharia D , Saini SK, Garg M A Hospital Based Prospective Study to Evaluate the Effect of Obesity on Cardiovascular Autonomic Functions in School Children . HECS Int J Comm Health Med Res 2020; 6(3)1-6.

INTRODUCTION

Obesity is a disorder with a multifactorial etiology resulting from a complex interaction between the environment, behaviour and genetic susceptibility.[1] As the prevalence of this disorder grows worldwide, obesity is increasingly considered a major public health problem.[2]

Obesity occurs when caloric intake exceeds energy expenditure and the excess calories are stored in an adipose tissue. Therefore, the ultimate cause of obesity is suggested to be an imbalance between energy intake and expenditure resulting from complex interaction of genetic, physiological, behavioural and environmental factors.[3]

The consequences of obesity in childhood and adolescence include arterial hypertension, atherosclerosis, dyslipidemia, diabetes, obstructive sleep apnea, alterations in the musculoskeletal system, depression and a reduction in quality of life.[4]

Since autonomic nervous system is involved in energy metabolism and regulation of cardiovascular system.[5] It is conceivable that one or more sub groups of obesity have an alteration in their autonomic nervous system that may promote obesity and account for several clinical consequences of obesity.

Several studies in literature suggest that autonomic nervous system of obese individuals is chronically altered. The activity of sympathetic nervous system is a determinant of energy

expenditure.[6] It has been observed that Individuals with low resting muscle sympathetic nerve activity may be at risk for body weight gain resulting from a lower metabolic rate. Obesity was found to be associated with decreased sympathetic activity in animal models.[7] Experimentally induced ventro-medial hypothalamic lesions resulted in decreased sympathetic activity, increased parasympathetic activity and obesity.[5] Overfeeding is found to be associated with sympathetic activation and there is evidence that adrenergic mechanisms contribute to cardiovascular complications.[8] So this study is done to investigate the cardiovascular autonomic nervous functions in obese school children.

MATERIAL & METHODS:

150 School children were randomly selected to obtain mixed group of children belonging to mixed socioeconomic status of age group 9-16 from various schools of Bikaner. Then they were screened to exclude underweight children by using lower cut-off limits of BMI. 50 underweight children were excluded and remaining 100 children were selected for the study. Now these 100 children were again segregated into two groups: Obese group (22) and Non obese group (78). This segregation was done using international obesity task force cutoffs because the IOTF group combined the childhood and adult definitions of overweight and obesity prevalent then, by taking at age 18 years, those percentiles that corresponded to the BMI's of 25 and 30 Kg/m^2, and using these same percentiles throughout the age range for specifying overweight and obesity in childhood in girls and boys separately.

Inclusion criteria:

1. Children with age ranging between 9 – 16 years of both the gender.
2. Physically and mentally fit.
3. Cooperative and capable of understanding the procedure.

Exclusion criteria:

1. Children suffering from medical ailments or anxious, apprehensive and uncooperative.
2. Any systemic illness that is likely to affect cardiovascular autonomic functions
3. Any major psychiatric illness.

Method:

Anthropometric measurements were done utilizing the standard equipments and methodology. Weight was recorded using spring weighing machine approximated to the nearest kilogram and height was recorded using the stadiometer attached to the wall to the nearest centimetre. All the measurements were done after removing shoes and all the loose outfits of the child.

Blood pressure (BP) was recorded from the left arm in supine position after giving adequate rest to the child measured with sphygmomanometer. Body mass index (BMI) was calculated as weight in kilograms / (Height in meter)^2. International Obesity Task Force (IOTF) classification was utilized for the estimation of obese subjects.

Procedure of autonomic evaluation:

The subject was asked to relax in supine position for 30 minutes. The resting heart rate was recorded on a standard ECG from lead II, at a paper speed of 25 mm/sec .The cardiovascular tests performed are detailed below in the order of execution. These tests were demonstrated to the subjects.

1. Deep breathing test or heart rate response to deep breathing or Expiration: inspiration ratio:

In the sitting position subject was asked to breathe quietly and deeply at the rate of 6 breaths per minute. A continuous ECG was recorded for six cycles with marker to indicate the onset of each inspiration and expiration. Variation in heart rate was calculated as rate of longest R-R interval during expiration to shortest R-R interval during inspiration.[9] A value of 1.20 or higher was taken as normal.[10]

2. Heart-Rate variation to Valsalva Manoeuver:

The subject was seated comfortably and was asked to blow into a mouthpiece connected to a mercury sphygmomanometer and holding it at a pressure of 40 mm of mercury for 15 seconds, while a continuous ECG was being recorded. The ECG was continued to be recorded after release of pressure at the end of 15 seconds for 30 seconds. The heart rate changes induced by the valsalva manoeuver was expressed as the ratio of the maximal tachycardia during the manoeuver to the maximal bradycardia after the manoeuver. This ratio was defined as the Valsalva ratio and was calculated as the ratio of maximum R-R interval after the manoeuver to minimum R-R interval during the manoeuver.[11]

Valsalva ratio(VR) = maximal tachycardia/maximum bradycardia= maximum R-R interval/minimum R-R interval.

A value of 1.10 or less is defined as an abnormal response, 1.11-1.20 as borderline, and 1.21 or more as a normal response.[12]

3. Heart rate response to standing - Postural Tachycardia Index (PTI) or 30:15 ratio:

The subjects were asked to lie on the examination table quietly while heart rate is being recorded on ECG. They were then asked to stand-up unaided and ECG was recorded for 1 minute. The shortest R-R interval at or around 15th beat and longest R-R interval at or around 30th beat was measured. The result was expressed as ratio of 30/15.

PTI= Longest R-R interval at 30th beat / shortest R-R at 15th beat. A ratio of 1.00 or less was defined as an abnormal response, 1.01-1.03 as borderline and 1.04 as normal response.[13]

4. Blood Pressure Response to Standing (Orthostatic test):

The subject was asked to rest in a supine position for 5 minutes. The resting BP was recorded. The subject was then asked to stand unaided and remain standing unsupported for 3 minutes. The BP was recorded at 30 seconds and 3 minutes after standing up. The difference between the resting and standing BP levels was calculated.

The fall in systolic BP at 30 seconds on standing noted. A fall of 30 mm Hg or more was defined as abnormal, fall between 11-29

mm Hg as borderline and fall of 10 mm Hg or less was considered normal.[14]

5. Blood Pressure Response to Sustained Handgrip:

In this test, sustained muscle contraction is measured by a handgrip dynamometer, causes a rise in systolic and diastolic blood pressure and heart rate. The dynamometer is first squeezed to isometric maximum, and then held at 30% maximum for 5 min. if possible, although even 3 minutes may be adequate. Blood pressure was recorded in the non exercising arm thrice at 1-minute interval during the procedure. The maximum reading of the diastolic blood pressure was taken as the final value. Then the rise in diastolic blood pressure was calculated by subtracting resting diastolic blood pressure from this value. A rise in DBP of less than 10 mm Hg was defined as abnormal, 11-15 mm Hg as borderline and 16 mm Hg or more as normal.[15]

Statistics:

The quantitative data was expressed as Mean ± S.D. and the student's 't' test was used to compare the differences between the respective means. All p values were 2 tailed, p value of <0.05 was considered significant.

RESULTS:

The table no. 1 shows BMI in obese and non-obese girls and boys. The comparison of mean value of parasympathetic and sympathetic activity in boys and girls shows in table no. 2.

Table 1: The mean value of BMI in obese and non-obese in boys and girls

	Boys		Girls	
	Non-obese	Obese	Non-obese	Obese
BMI (kg/m^2)	18.12±1.802	25.96±4.219	17.65±1.82	24.12±4.83

Table 2: The comparison of mean value of Autonomic function test in boys and girls

Autonomic function test	Boys			Girls		
	Normal	Abnormal	p-value	Normal	Abnormal	p-value
30:15 ratio	1.393±0.176	1.398±0.265	>0.05	1.379±0.20	1.323±0.117	>0.05
Valsalva ratio	1.63±0.295	1.67±0.425	>0.05	1.68±0.265	1.51±0.252	>0.05
E-I ratio	1.49±0.53	1.40±0.219	<0.05*	1.487±0.182	1.34±0.148	<0.05*
Systolic BP lying to standing	6.05±2.77	13.17±4.33	<0.0001	6.23±2.79	11.77±3.76	0.0001
Diastolic BP Hand grip test	13.97±1.98	8.25±2.12	0.0001	13.56±2.49	8.36±1.88	0.0001
Resting systolic BP	108.5±9.78	114.27±10.12	<0.05	110.48±11.66	118.21±7.13	<0.05*
Resting diastolic BP	64.89±6.88	66.78±6.53	>0.05	63.07±5.89	67.68±6.52	<0.05*

DISCUSSION:

The result of our study shows reduced E:I (Expiration: Inspiration) ratio in obese children as compared to the normal children. A reduction in parasympathetic activity among obese children has also been reported by other authors. Our findings are consistent with Yakinci et al,[16] who observed the hypoactivity of parasympathetic nervous system, implying parasympathetic nervous system dysfunction as a risk factor or associated finding in childhood obesity, while investigating the autonomic nervous system function in childhood (7-13 years of age) obesity.

Rossi et al,[17] supports our results who reported a lower parasympathetic function in obese subjects. The finding was suggestive of a causal role of parasympathetic tone in sudden death. Our results corroborate with Hirsch et al,[18] who described an inverse relationship between amount of weight gain and lower

parasympathetic drive. The limited studies conducted in obese children indicate parasympathetic withdrawal and sympathetic predominance with metabolic changes such as impaired blood lipid profile. Arrone et al,[19] suggested that the ANS of individuals with obesity is chronically altered in a way that would tend to oppose their excessive adiposity, and that these autonomic changes are more likely to be responses to other forces that induce obesity, rather than being primary agents in the production of the disease.

Our results corroborate with Nagai et al,[20] who evaluated that obese children possess reduced sympathetic as well as parasympathetic nerve activities. He concluded that autonomic depression, which is associated with the duration of obesity, could be a physiological factor promoting the state and development of obesity.

Our findings are in line with Vanderlei et al,[21] who concluded that the obese children exhibited modifications in heart rate variability, characterized by a reduction in both sympathetic and parasympathetic activity. These findings stress the need for the early holistic care of obese children to avoid future complications.

The possible mechanisms are:

1. The hypothalamus is a regulatory centre of satiety and of the ANS. Therefore, abnormalities in the hypothalamus may cause obesity and autonomic dysfunction.[22] This may explain the alterations observed in the heart rate variability indices.
2. The exact mechanism that may cause impairment of parasympathetic nerve function has not yet been clearly established. Some researchers suggested that gradual development of insulin resistance in target tissues with the beginning of excess weight gain in obesity is responsible for subsequent development of hyperinsulinaemia. This hyperinsulinaemia has got a role in low cardiac vagal activity in obese person. Though the relationship between insulin resistance and parasympathetic dysfunction is not clear, but several researchers made various suggestions such as high insulin level or insulin resistance may cause damage to autonomic nerves at any level of their reflex arc, insulin resistance may cause a deterioration of microcirculation in many tissues including nerves which may lead to neural ischemia and thereby damage of cardiac parasympathetic nerve terminals occur at the level of cardiac muscle or vascular wall.[23,24]
3. Valensi et al,[6] observed cardiac parasympathetic dysfunction present in the obese subjects could be associated with higher carbohydrate intake and lower fat and protein intake which result in parasympathetic abnormality.
4. A reduction in vagal activity is associated with an increased risk for all-cause morbidity and mortality and for the development of several risk factors. Therefore, the reduction observed in obese children may be an early sign for the prediction of the risk for cardiovascular and metabolic disease.
5. In cardiac autonomic neuropathy, the disruption of parasympathetic nervous system is usually detected earlier than that of the sympathetic nervous system. Decrease in heart rate variability is noticed as first indicator of cardiac neuropathy and decrease in Expiration: Inspiration ratio is considered to be a sign of parasympathetic dysfunction.[25]
6. Our result is also supported by Rissanen et al,[26] who documented that cardiac parasympathetic activity increases with weight loss in obese women. This increase may not be maintained long term if body weight is regained. The rise of cardiac parasympathetic activity is correlated with decrease of body fat mass, abdominal fat, serum insulin, and heart rate. Cardiac parasympathetic activity is not related to resting energy expenditure.

SYMPATHETIC REACTIVITY

The results of our study indicates higher baseline diastolic blood pressure and systolic blood pressure prior to isometric handgrip exercise and blood pressure response on lying to standing in obese group children which shows the reduced sympathetic activity in obese children .The result of our study is in line with Kalpana et al,[27] showed the diastolic pressure was significantly higher at rest, but showed reduced rise during handgrip test in comparison with normal weight individuals. Also, the rise in diastolic pressure exhibited a negative relation with BMI.

Grewal and Gupta[28], also supported our study who found that the mean values of hand grip response were significantly lower in study group as compared with the controls.

Guizar et al[29] who have observed increased blood pressure levels in obese children and adolescents respectively as compared to their control counterparts.

Our findings are in consistent with Akhter et al,[30] who conducted a cross sectional study which showed that mean values of resting heart rate, resting systolic and diastolic blood pressure were significantly ($p<0.05$) higher and both the sympathetic nerve function parameters (blood pressure response to hand grip and blood pressure response to standing) were significantly ($p>0.05$) lower in obese compared to those of non obese control subjects.

Our study is also in line with Bedi et al,[31] who conducted autonomic function tests in 30 normal and 30 obese children aged between 5 and 10 years and concluded that there is compromised autonomic nervous system functions in the obese group compared with controls.

Our results corroborate with Rajalakshmi et al,[32] who observed over weight and obese young adults had elevated resting blood pressure and showed increased response to steady exercise which could be due to alterations in the autonomic activity in obese persons.

Study of Tonhajzerova I et al,[33] is also consistent with our study who found a significantly higher resting SBP and DBP in Mexican adolescent boys and Slovakian adolescents, respectively.

The first effect is due to increased activity in cardiac sympathetic fibers and second due to increased activity in peripheral vasoconstrictor fibers. Such preferential activation of peripheral vasoconstrictor fibers has been attributed to cause cold induced vasoconstrictor response in normotensive population[34,35] and in hypertensive.[36] The isometric exercise induced increase in heart rate, cardiac output and blood pressure, reported in the literature[37] can be explained on the basis of activity in the cardiac sympathetic fibers. The obese children showed truncated response in cardiac sympathetic activity resulting in borderline response to isometric exercise.

The results of present study suggest that obese children have autonomic dysfunctions characterized by a reduction of both parasympathetic and sympathetic activity. The continuing increase in the number of obese children is alarming due to potential risk of premature health problems. Moreover, obese children have a high likelihood of becoming obese adults, and obese adults who were once obese children have lower treatment response than those who become obese in adulthood. These concerns about the effects of obesity reinforce the need for the prevention and treatment of the condition in childhood. The

importance of changes in the lifestyle of these children must be emphasized, especially with regard to eating habits and the practise of regular physical activity.

CONCLUSION:

We concluded that parents of such obese children should take this very seriously and must guide their kids to go for physical exercise as well as diet control to check the rapidly increasing weight.

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