Harsukh Educational Charitable Society
International Journal of Community Health and Medical Research
Journal home page: www.ijchmr.com doi: 10.21276/ijchmr
Official Publication of "*Harsukh Educational Charitable Society*" [Regd.]
ISSN E: 2457-0117 **ISSN P:**2581-5040 **RNI No. -** PUNENG/2017/75049 **Index Copernicus value 2016 =** 52.13

Original Article

A COMPARATIVE STUDY OF SYMPATHETIC AUTONOMIC FUNCTION INDICES AMONG OBESE AND NON OBESE INDIVIDUALS OF WESTERN RAJASTHAN

Kirti Goyal[1] , Prahlad Kalwan[2]

[1] PhD Scholar, Department of Physiology, [2] Senior Resident, Department of Surgery Dr S.N. Medical College, Jodhpur, Rajasthan

ABSTRACT
Background:-Obesity is one of the most common significant health hazards and is associated with autonomic dysfunction. **Material and method:-** In the present study, 40 obese subjects (body Mass Index >25 kg/m2) and 40 non-obese subjects (Body Mass Index <25kg/m2) were selected to study the effect of obesity on the sympathetic nervous system. To test the functioning of sympathetic nervous system in these groups, two non-invasive cardiovascular autonomic function tests -blood pressure response to standing and blood pressure response to sustained handgrip were performed. **Results:-** Result of both test were lower in obese subjects as compared to non obese subjects. But result of only sustained grip handgrip exercise shows significant result. **Conclusion:-**Thus, obesity is associated with altered sympathetic nervous system which may result in various cardiovascular complications.

Keywords: Obesity, Autonomic Dysfunction, Body Mass Index, Cardiovascular Autonomic Function Test, Blood Pressure Response To Standing And Blood Pressure Response To Sustained Handgrip.

Corresponding Author: Dr.Prahlad Kalwan, Senior Resident, Department of Surgery, Dr S.N. Medical College, Jodhpur, Rajasthan

This article may be cited as: Goyal K and Kalwan P. A Comparative Study Of Sympathetic Autonomic Function Indices Among Obese And Non Obese Individuals Of Western Rajasthan. HECS Int J Comm Health Med Res 2018; 4(4):1-4

INTRODUCTION

Obesity is a global problem, prevalence of which is in the rise in nearly every country because of physical inactivity. Health implications of obesity are becoming more evident in children and adolescents. Autonomic nerve dysfunction may co exist with few recognized complications such as cardiovascular disorders in overweight and obese person. Autonomic nervous system is a centre for the coordination of different body system.[1] The energy balance is to a reasonably large extent affected by the status of autonomic nervous system (ANS) activity in the individual. It has been proposed that there may be a reduced reactivity in established obesity, which contributes to maintenance of the obese state.[2] In terms of metabolism, the sympathetic nervous system is fundamental in controlling daily energy expenditure via the regulation of resting metabolic rate and initiation of thermogenesis in response to physiologically relevant stimuli, that is, changing energy states, food intake, carbohydrate consumption, hyperinsulinemia, and exposure to cold. Activation of sympathetic nerves innervating the liver, pancreas, skeletal muscle, and adipose tissue can also elicit acute catabolic responses (i.e., glycogenolysis and lipolysis). It is important to note that not all organs are targeted equally by the sympathetic nervous system with the metabolic effects ensuing from increased central sympathetic outflow dependent on the adrenergic receptors present in the target organ, the number of neurons recruited, and whether an individual is in a fasted or postprandial state. Cardiovascular autonomic function tests (CAFTs) assess sympathetic nervous system activities. These non□invasive set of tests are easily administrable in small clinical settings. Body mass index (BMI) is a simple, valid and inexpensive surrogate measure of obesity suggested by the World Health Organization (WHO).[3]Screening of obesity by BMI helps to stratify people with future health risks.With this background,our study was aimed at assessing the extent of SNS dysfunction tested by CAFT in healthy subjects whose BMI is more than 25kg/m^2.

MATERIAL AND METHODS

A total 80 healthy subjects were included in this study with age range from 18 to 60 years. The study was conducted in the

Department of Physiology at Dr. S. N. Medical College, Jodhpur. The approval of the Ethical Committee was obtained.

Inclusion Criteria: Only healthy subjects of western Rajasthan were included in this study. Based on the BMI they were divided into two groups, Study Group, composed of 40 obese individuals, and Control Group, composed of 40 non obese individuals, matched for age and height.

Exclusion Criteria: The non smoker, non alcoholic, non diabetic, having normal pulse rate, blood pressure, normal heart sounds and having no evidence of illness and having perfect physical, mental and psychological well being were included in the study.

Obesity

Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health.[4]

Weight in kilograms and height in centimeters were measured and recorded for all the subjects to calculate the body mass index (BMI) by **Quetelet's index** as follows:

$$\textbf{BMI=Weight(kg)/(Height(m))}^2$$

A person with a BMI of 30 or more is generally considered obese. A person with a BMI equal to or more than 25 is considered overweight.[5] In our study both overweight and obese subjects to be clubbed together in Study group and identical number of age and sex matched non-obese subjects served as controls.

A brief history was taken and general physical examination of all the volunteers was done with main emphasis on cardiovascular diseases, renal diseases. None of the subjects took any medication at the time of study. All the tests were carried out between 11 am to 4 pm. The procedure was explained and informed consent was obtained after the subjects had read a description of the experimental protocol.

Test of predominantly sympathetic function are:-

1. **Blood - pressure response to standing**

The BP of the subject was recorded at lying down and again when the subject stands up from supine position.

Stimulus - Change of posture from lying to standing
Afferents - Baroreceptors & Cranial nerve ninth and tenth
Efferents - Sympathetic (adrenergic),Parasympathetic (Cardiovagal, Cholinergic)
Normal response - Initially increase in heart rate followed by decrease in heart rate and fall in blood-pressure.

In normal subjects systolic BP does not fall by more than 10 mm Hg and in autonomic dysfunction it falls by >20-30 mm Hg. Orthostatic hypotension was defined as a fall of ≥20 mm Hg in systolic and /or ≥ 10 mm Hg in diastolic BP from lying to standing position.[6]

Values of orthostatic fall in systolic BP:-

a) Normal ≤10 mmHg
b) Borderline 11-29 mmHg
c) Abnormal ≥30 mmHg

2. **Blood –pressure response to Sustained Handgrip (SHG)**

This test studies the blood pressure response to an isometric handgrip exercise. Initially the subject was asked to exert maximal hand grip strength on hand grip dynamometer with dominant hand. First the maximum voluntary contraction (MVC) (MAXIMAL ISOMETRIC TENSION i.e. T_{max}) is determined and then the subjects were asked to press the handgrip dynamometer for at 30% of maximal voluntary effort. The BP was recorded in contra lateral arm and rise in diastolic BP was measured.

Stimulus - Isometric exercise
Afferents-Myelinated mechanosensitive & unmyelinated chemosensitive afferent from muscles
Efferents - Sympathetic (adrenergic)
Normal response - Increase in systolic & diastolic BP, increase in heart rate

Value of Rise in Diastolic BP after sustained handgrip exercise [Ewing and Clarke grading][7]:-

a) Normal ≥16 mmHg
b) Borderline 11-15 mmHg
c) Abnormal <10 mmHg

Analysis of data

Collected data were entered in computer based Microsoft Excel sheet. Comparisons were done by applying Student's 't' test .

RESULTS

Table 1 : Anthropometric variables.

PARAMETER	NON OBESE SUBJECTS		OBESE SUBJECTS		P VALUE
	MEAN	S.D.	MEAN	S.D.	
AGE (years)	37.6	11.5	40.0	10.4	0.2947 (NS)
WEIGHT (kg)	60.26	6.56	81.22	10.48	<0.0001 (HS)
HEIGHT (m)	1.67	5.63	1.69	6.4	0.03(S)
BMI (kg/m^2)	21.54	1.99	28.01	2.02	<0.0001 (HS)

Non obese subjects having BMI between18.50 to 24.90 kg/m^2, Obese subjects having BMI >25 kg/m^2

NS= not significant(p value >0.05), S=Significant(p value <0.05), HS= significant at <0.0001 level.
All values are expressed as Mean and Standard deviation (S.D.).

Table 2: Sympathetic function test in Non obese and obese subjects.

PARAMETER	NON OBESE SUBJECTS		OBESE SUBJECTS		P VALUE
	MEAN	S.D.	MEAN	S.D.	
FALL IN SBP(mm Hg)	3.65	6.52	0.03	6.83	0.7732(NS)
RISE IN DBP(mm Hg)	13.05	5.84	12.4	12.51	<0.0001(HS)

NS= not significant(p value >0.05), HS= Highly Significant(p value <0.0001),All data are expressed as Mean and Standard deviation(S.D.)

DISCUSSION

The present study was carried out in 80 healthy subjects within the age range of 21-60 years, to assess the influence of obesity on sympathetic nervous system function. The subjects were distributed into two groups- Obese (body Mass Index >25 kg/m^2) and non obese group (body Mass Index <25 kg/m^2). Total 40 subjects (50%) were included in each group. Two non-invasive cardiovascular sympathetic function tests -blood pressure response to standing and blood pressure response to sustained handgrip were performed on subjects. In the present study, blood pressure response to standing was insignificantly (p>0.05) lower in obese subjects 0.03±6.83 than 3.65±6.52 than in non obese subjects. In our study, blood pressure response to sustained handgrip exercise showed statistically highly significant (p<0.0001) decrease in obese subjects 12.4±12.51 as compared to non-obese subjects 13.05±5.84. This decreased sympathetic activity may be the result of a defect in sympathetic nerve activation or alternatively in peripheral adrenoreceptors behavior. Similar results have been reported by other researchers- Garg R et al[8] suggested that there was decreased in blood pressure response to isometric handgrip exercise test in the obese people in contrast to the control group. It shows the decreased activity of the sympathetic nervous system or to a lower increase in peripheral resistance to manoeuvres activating sympathetic system.
Valensi et al[9] and Bedi M et al[2] have also demonstrated sympathetic insufficiency in obese state. Valensi et al showed that glucose induced inhibition of the lipid oxidation rate in obese women is greater in the patients with autonomic dysfunction. This could be because of decrease in sympathetic activity.
The hand grip and cold pressor tests show reactivity to stress in conditions of isometric exercises and cold stress,respectively. It has been proposed that there may be reduced reactivity in established obesity which contributes to maintenance of the obese state[10]. In a remarkable paper by Peterson et al[11], suggested that decrease in sympathetic activity causes a disordered homeostatic mechanism which promotes excessive energy storage Similarly Nargai et al in his study found evidence of autonomic depression in obese children which was associated with duration of obesity[12].
The above studies, as well as other reports, indicated that a reduced absolute sympathetic activity or a blunted sympathetic reactivity in certain physiological conditions was found in adult obese subjects.[13-17] Our result are not in agreement with study done by N Shetty et al[18], Yakinci et al[19] , Schwartz RS et al[20], Sowers JR et al[21] and Boehringer K et al[22].

CONCLUSION

Fom our study it is concluded that obesity is associated with sympathetic nervous system dysfunction which may result in various cardiovascular complications. So, if this dysfunction is diagnosed early by doing various autonomic function tests, it will be of great help in identification of those which are prone to weight gain and at risk of various cardiovascular complications.

ACKNOWLEDGEMENTS

I am very grateful and would sincerely thank to the faculty members and staff of the Department of
Physiology, Dr. S. N. Medical College and Hospital, Jodhpur, Rajasthan, India, for their immense cooperation
and support.

REFERENCES

1. Bray GA. Autonomic and endocrine balance in the regulation of energy balance. Fed Proc 1986;45:1404-10.
2. Bedi M, Khullar S, Varshney VP. Assessment of autonomic function activity in obese children. Vasc Dis Prev 2009;6:139-41.
3. Hall JE. Dietary balances; regulation of feeding; obesity and starvation; vitamins and minerals. In: Guyton and Hall Textbook of Medical Physiology. 12th ed. Philadelphia: Saunders, an Imprint of Elsevier Inc.; 2011. p. 850.
4. Eikelis N., Schlaich M, Aggarwal A, Kaye D, Esler M. Interactions between leptin and the human sympathetic nervous system. Hypertension 2003; 41:1072-1079.
5. Haynes WG, Morgan DA, Walsh SA, Mark AL, Sivitz WI. Receptor-mediated regional sympathetic nerve activation by leptin. J Clin Invest 1997; 100:270-278.
6. Consensus committee. The definition of orthostatic hypotension, pure autonomic failure and multiple system atrophy.J Auton Nerv Syst 1996;58(1-2):123-4
7. Ewing DJ , Irving JB, Kerr F, Wildsmith JAW, Clarke BF. Cardivascular responses to sustained handgrip in normal subjects and in patients with diabetes mellitus: A test of autonomic function Clin Sci Mol Med. 1974;46:295-306.
8. Rinku Garg, Varun Malhotra, Neera Goel, Usha Dhar, Yogesh Tripathi. A study of autonomic function tests in

obese people. Int J Med Res Health Sci. 2013;2(4):750-755.
9. Valensi P, Lormeau B, Dabbech M, et al. Glucose induced thermogenesis , inhibition of lipid oxidation rate and autonomic dysfunction in non-diabetic obese women. Int J Obes Relat Metab Disord 1998; 22(6): 494 9.
10. Baeks MA Van. The peripheral sympathetic nervous system in human obesity. Obes Rev 2001; 2(1): 3-14.
11. 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.
12. Nagai N, Matsumoto T, Kita H, Moritani T. Autonomic nervous system activity and the state and development of obesity in Japanese school children. Obes Res 2003;11:25□32.
13. Acheson KJ, Ravussin E, Schoeller DA, Christin L, Bourquin L, Baertschi P, Danforth Jr E, Jequier E. Two-week stimulation or blockade of the sympathetic nervous system in man: influence on body weight, body composition, and twenty-four-hour energy expenditure. Metabolism, 1988; 37: 91–98.
14. Buemann B, Astrup A, Madsen J, Christensen NJ. A 24-h energy expenditure study on reduced-obese and nonobese women: effect of beta-blockade. Am J Clin Nutr, 1992; 56: 662–670.
15. Toubro S, Sorensen TIA, Ronn B, Christensen NJ, Astrup A. Twenty-four-hour energy expenditure: the role of body composition, thyroid status, sympathetic activity, and family membership.
J Clin Endocrinol Metab, 1996; 81: 2670–2674.
16. Matsumoto T, Miyawaki C, Ue T, Kanda T, Yoshitake Y, Moritani T. Comparison of thermogenetic sympathetic response to food intake between obese and non-obese young women. Obes Res, 2001; 9: 78–85.
17. Petretta M, Bonaduce D, de Filippo E, Mureddu GF, Scalfi L, Marciano F, Bianchi V, Salemme L, de Simone G, Contaldo F. Assessment of cardiac autonomic control by heart period variability in patients with early onset familial obesity. Eur J Clin Invest, 1995; 25: 826–832.
18. Shetty S, Parakandy SG, Nagaraja S. Cardiac autonomic function tests in overweight adolescents. Indian J Basic Appl Med Res 2015;1:316□20.
19. Yakinci C, Mungen B, Karabiber H, Tayfun M, Evereklioglu C. Autonomic nervous system functions in obese children. Brain Dev 2000;22:151□3.
20. Schwartz RS, Halter JB, Bierman EL: Reduced thermic effect of feeding in obesity: Role of norepinephrine. Metabolism 198332:114-117.
21. Sowers JR, Whitfield LA, Catania RA, Stern N, Tuck ML, Dornfield L, Maxwell M: Role of the sympathetic nervous system in blood pressure maintenance in obesity. Clin Endocrinol Metab 1982^4:1181-1186.
22. Boehringer K, Beretta-Piccoli C, Weidmann P, Meier A, Ziegler W: Pressor factors and cardiovascular pressor responsiveness in lean and overweight normal or hypertensive subjects. Hypertension 1982;4:697-702.

Source of support: Nil **Conflict of interest:** None declared