

## A STUDY OF AUTONOMIC FUNCTION TESTS IN OBESE SUBJECTS

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2023; 5 (5); 936-939**Abstract**

**Background:** Obesity prevalence is soaring in industrialized countries and progressively increasing in the developing world. This has been called “New world syndrome” and is a reflection of massive social, economic and cultural problems currently facing the developing and developed countries. **Objective:** to study the autonomic function tests in obese subjects. **Material & Methods:** The present study was carried out in 60 obese subjects (30 males and 30 females) and 60 non-obese subjects (30 males and 30 females). The subjects were selected from the general population of town area. **Results:** The subjects were selected from general population of town area. 60 obese subjects (mean age  $36.03 \pm 6.65$  years and mean BMI  $32.74 \pm 2.07$  kg/m<sup>2</sup>) and 60 normal weight subjects (mean age  $36.12 \pm 6.53$  years and mean BMI  $22.43 \pm 1.75$  kg/m<sup>2</sup>) were studied. **Conclusion:** Obesity is associated with both sympathetic and parasympathetic nervous system dysfunctions which may result in various cardiovascular complications.

**INTRODUCTION**

Altered patterns of nutrition and reduction in work-related energy expenditure, which leads to sedentary life style, have led to obesity becoming a truly global health issue.<sup>[1]</sup> Obese people have greater risk of developing both cardiovascular and metabolic diseases like hypertension, atherosclerosis, diabetes mellitus and gallbladder diseases.<sup>[3]</sup>

In the mid-1990s WHO expressed concern to the growing obesity epidemic throughout the world. According to world health statistic report of 2012, prevalence of obese subjects of >20 years of age is 1.3 in males and 2.5 in females in India.<sup>[4]</sup> Overweight population will reach approximately to 2.3 billion and obese population to 700 million by 2015.<sup>[5]</sup>

Obesity is a condition of excess fat deposition in the body.<sup>[6]</sup> 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.<sup>[2]</sup>

A regulatory system that maintains constant energy storage is likely to involve complex interactions among humoral, neural, metabolic, and psychological factors. It has been suggested that the Autonomic Nervous System (ANS) acts as central in

the co-ordination of this system.<sup>[7]</sup> Overfeeding is found to be associated with sympathetic activation and there is evidence that adrenergic mechanisms contribute to cardiovascular complications.<sup>[2]</sup> The sympathetic nervous system (SNS) plays an essential role in the regulation of metabolic and cardiovascular homeostasis. Low SNS activity has been suggested to be a risk factor for weight gain and obesity development.<sup>8</sup> With a 10% increase in body weight above the usual or starting weight, there was a decline in parasympathetic power accompanied by a rise in mean heart rate.<sup>[9]</sup>

Hypothalamus plays an important role in the regulation of body weight. The hypothalamus and related parts of the brain play a key role in the regulation of food intake. Many years ago, Sherrington called the hypothalamus “the head ganglion of the autonomic system”.<sup>[2]</sup>

Previous studies on autonomic nervous activity in obese persons have reported controversial findings; these include hypo activity of parasympathetic and associated hyperactivity of sympathetic nerve function,<sup>[10]</sup> both reduced sympathetic and parasympathetic nerve activity,<sup>[11]</sup> and increased parasympathetic activity with a decreased sympathetic nerve activity.<sup>[12]</sup> Sometimes, obesity is associated with minor impairments of autonomic nerve function and in most of the cases, changes were found in parasympathetic division of autonomic nervous system which is analogous to the

early stage of autonomic neuropathy in diabetes mellitus.<sup>[13]</sup> Obesity related health problems including various cardiovascular and metabolic diseases are not uncommon in our community. Since the autonomic nervous system is involved in energy metabolism and the regulation of the cardiovascular system, it is conceivable that one or more subgroups of persons with idiopathic obesity may have an alteration in their autonomic nervous systems. It accounts for several clinical consequences of obesity or protect against certain health problems, such as sudden death or hypertension. Hence, in the present research work, we have studied autonomic function tests in obese subjects.

## MATERIALS AND METHODS

**The following parameters of autonomic function tests were recorded in all the subjects:**

1. Heart rate (beats/minute).
2. Resting systemic arterial blood pressure (mmHg)

### Systolic blood pressure

- Diastolic blood pressure

### Parasympathetic Function Tests

1. Heart rate during deep breathing (expiration / inspiration ratio).
2. Heart rate response to valsalvamanoeuvre (for 15 seconds).
3. Heart rate response to immediate standing from supine position (30:15ratio).
4. Standing to lying ratio (S/L ratio) (Heart rate response to standing and lying position).

### Sympathetic Function Tests

1. Orthostatic test (blood pressure response to standing from the supine position).
2. Blood pressure response to sustained hand-grip test.
3. Blood pressure response to cold pressor test (temperature was maintained at 4-6°C).

1. **Heart rate (beats/minute)**

2. Heart rate during deep breathing (expiration / inspiration ratio).<sup>[6]</sup>
3. Heart rate response to valsalvamanoeuvre (for 15 seconds).<sup>[6]</sup>
4. Heart rate response to immediate standing (30:15 ratio).<sup>[6]</sup>
5. Standing to lying ratio (S/L ratio) (heart rate response to standing and lying position).<sup>[6]</sup>
6. Resting systemic arterial blood pressure (mmHg).<sup>[14]</sup>
7. Orthostatic test (blood pressure response to standing from the supine position).<sup>[6]</sup>
8. Blood pressure response to sustained hand-grip test.<sup>[14]</sup>
9. Blood pressure response to cold pressortest.<sup>[6]</sup>

### Statistical Analysis.<sup>[15]</sup>

For statistical analysis, we divided subjects in two groups- obese group (including males and females) and non-obese group (including males and females). All the data was presented as mean  $\pm$  S.D. (Standard Deviation). To study the significance of difference in parameters between obese and non-obese groups, we applied the student's unpaired 't' test and calculated the 'p' value by using Graphpad prism 5.0 software. The results were expressed as statistically non significant if  $p > 0.05$ , as statistically significant if  $p < 0.05$ , as highly statistically significant if  $p < 0.001$ .

## RESULTS

The difference in mean body mass index between obese and non-obese subjects is statistically highly significant ( $p < 0.0001$ ). The resting heart rate is insignificantly higher ( $p > 0.05$ ) in obese subjects as compared to non-obese subjects. The heart rate response to deep breathing (expiration/inspiration ratio) is lower in obese subjects as compared to non-obese subjects, which is statistically highly significant ( $p < 0.0001$ ). The heart rate response to valsalva maneuver (Valsalva ratio) is more in obese subjects as compared to non-obese subjects, which is statistically insignificant ( $p > 0.05$ ).

**Table1: Comparison of heart rate response to valsalva maneuver (Valsalva ratio) between obese and non-obese subjects**

Group	Heart rate response to valsalva maneuver (Valsalva ratio) Mean $\pm$ S.D.	t value	p value
Obese (n=60)	1.23 $\pm$ 0.12	0.85	0.39
Non-obese (n=60)	1.22 $\pm$ 0.07		

The heart rate response to immediate standing (30:15 ratio) is decreased in obese subjects as compared to non-obese subjects, which is statistically insignificant ( $p > 0.05$ ).

**Table 2: Comparison of heart rate response to immediate standing (30:15 ratio) between obese and non-obese subjects**

Group	Heart rate response to immediate standing (30:15 ratio) Mean $\pm$ S.D.	t value	p value
Obese (n=60)	1.05 $\pm$ 0.12	0.41	0.68
Non-obese (n=60)	1.06 $\pm$ 0.05		

The heart rate response to standing and lying i.e. standing to lying ratio (S/L ratio) is decreased in obese subjects as compared to non-obese subjects, which is statistically significant ( $p < 0.01$ ).

Table 3: Comparison of heart rate response to standing and lying (S/L ratio) between obese and non-obese subjects

Group	Heart rate response to standing and lying(S/L ratio) Mean± S.D.	tvalue	pvalue
Obese(n=60)	0.99±0.13	2.73	0.007
Non-obese(n=60)	1.04 ±0.07		

The resting systolic blood pressure (mmHg) is more in obese subjects as compared to non-obese subjects, which is statistically highly significant ( $p < 0.0001$ ).

Table 4: Comparison of resting systolic blood pressure between obese and non-obese subjects

Group	Resting systolic BP(mmHg) Mean± S.D.	tvalue	pvalue
Obese(n=60)	124.3±5.19	5.9	<0.0001
Non-obese(n=60)	119.6±3.25		

The resting diastolic blood pressure (mmHg) is significantly higher ( $p < 0.001$ ) in obese subjects as compared to non-obese subjects. There is fall in systolic blood pressure (mmHg) on standing from supine position in both the groups. The fall in mean systolic blood pressure on standing from supine position is significantly more ( $p < 0.001$ ) in obese subjects as compared to that in non-obese subjects. The diastolic blood pressure response (mmHg) to orthostatic test shows more change in blood pressure to standing from supine position in obese as compared to non-obese subjects, but the change is statistically insignificant ( $p > 0.05$ ). The systolic blood pressure response (mmHg) to sustained hand grip test shows less increase in blood pressure after the test in obese subjects as compared to non-obese subjects, which is statistically highly significant ( $p < 0.0001$ ). The diastolic blood pressure response (mmHg) to sustained hand grip test shows less increase in blood pressure after the test in obese subjects as compared to non-obese subjects, which is statistically significant ( $p < 0.05$ ). The systolic blood pressure response (mmHg) to cold pressor test shows increase in blood pressure after test is less in obese subjects as compared to non-obese subjects, which is statistically highly significant ( $p < 0.0001$ ). The diastolic blood pressure response (mmHg) to cold pressor test shows increase in blood pressure after test is more in obese as compared to non-obese subjects, which is statistically insignificant ( $p > 0.05$ ).

## DISCUSSION

In the present research, a careful statistical analysis of the autonomic function status in obese and non-obese subjects was done. The mean age of obese subjects was  $36.03 \pm 6.65$  years and that of non-obese subjects was  $36.12 \pm 6.53$  years. The mean body mass index of obese subjects was  $32.75 \pm 2.02$  kg/m<sup>2</sup> and that of non-obese subjects was  $22.44 \pm 1.78$  kg/m<sup>2</sup>. The age difference between obese and non-obese subjects was not statistically significant

( $p > 0.05$ ), whereas, the BMI difference between obese and non-obese subjects was statistically highly significant ( $p < 0.0001$ ).

Studies on autonomic function in obesity are scanty, we analyzed comparison of parasympathetic function tests and sympathetic function tests between obese and non-obese subjects:

### Parasympathetic function tests

In the present study, the resting heart rate (beats/minute) was insignificantly ( $p > 0.05$ ) higher in obese subjects ( $74.93 \pm 8.33$ ) as compared to non-obese subjects ( $74.57 \pm 9.07$ )

Rossi M et al (1989)<sup>[13]</sup>, Plaengdee J et al (2009),<sup>[16]</sup> found that mean heart rate (beats/minute) was significantly higher in obese subjects as compared to non-obese subjects.

Alvarez GE et al (2002)<sup>[17]</sup> found that the resting heart rate (beats/minute) was insignificantly higher in the lower abdominal visceral fat (LAVF) group as compared to higher abdominal visceral fat (HAVF) group.

In our study, heart rate response to deep breathing i.e. expiration/inspiration ratio showed statistically highly significant ( $p < 0.0001$ ) decrease in obese subjects ( $1.13 \pm 0.12$ ) as compared to non-obese subjects ( $1.25 \pm 0.08$ ).

Colak R et al (2000),<sup>[7]</sup> Grewal S et al (2011),<sup>[6]</sup> found that heart rate response to deep breathing was insignificantly decreased in obese subjects as compared to non-obese subjects. We found that heart rate response to valsalvamanuever i.e. valsalva ratio was higher in obese subjects ( $1.23 \pm 0.12$ ) than non-obese subjects ( $1.22 \pm 0.07$ ) and that was statistically insignificant ( $p > 0.05$ )

Rossi M et al (1989),<sup>[13]</sup> Colak R et al (2000),<sup>[7]</sup> found that valsalva ratio was insignificantly lower in obese subjects as compared to non-obese

Our study showed heart rate response to immediate standing i.e. 30:15 ratio was less in obese subjects ( $1.05 \pm 0.12$ ) than non-obese subjects ( $1.06 \pm 0.05$ ) and that was statistically insignificant ( $p > 0.05$ ) (Table no.5).

Rossi M et al (1989),<sup>[13]</sup> Garg R et al (2013),<sup>[18]</sup> found that heart rate response to standing (30:15 ratio) was significantly lower in obese subjects as compared to non-obese subjects.

#### **Sympathetic function tests**

In present study, the systolic blood pressure was more in obese subjects ( $124.3 \pm 5.19$  mmHg) as compared to non-obese subjects ( $119.6 \pm 3.25$  mmHg) and the diastolic blood pressure was also more in obese subjects ( $80.00 \pm 3.51$  mmHg) as compared to non-obese subjects ( $77.47 \pm 3.82$  mmHg), which were statistically highly significant ( $p < 0.0001$ ).

Grassi G et al (1994),<sup>[19]</sup> found that systolic and diastolic blood pressures insignificantly less in obese subjects.

In the present study, the systolic blood pressure response (mmHg) to standing from the supine position showed more fall in obese subjects ( $6.57 \pm 3.32$ ) as compared to non-obese subjects ( $4.90 \pm 2.69$ ), which was statistically significant ( $p < 0.001$ ).

Our finding was consistent with the findings of Colak R et al (2000),<sup>[7]</sup>

Our study showed less rise in the systolic blood pressure response (mmHg) to sustained hand grip test in obese subjects ( $11 \pm 2.53$ ) as compared to non-obese subjects ( $14.87 \pm 4.36$ ), which was statistically highly significant ( $p < 0.0001$ )

Sarwari K et al (2011)<sup>[20]</sup> found that rise in the systolic blood pressure (mmHg) to sustained hand grip test was insignificantly less in obese subjects as compared to non-obese subjects.

In our study, the diastolic blood pressure response (mmHg) to sustained hand grip test showed less rise in obese subjects ( $9.73 \pm 2.31$ ) as compared to non-obese subjects ( $10.9 \pm 2.79$ ), which was statistically significant ( $p < 0.05$ )

Rossi M et al (1988),<sup>[13]</sup> found that rise in diastolic blood pressure (mmHg) in sustained hand grip test was statistically insignificant in obese subjects as compared to non-obese subjects.

Colak R et al (2000),<sup>[7]</sup> found that change in diastolic blood pressure (mmHg) in sustained hand grip test was more and statistically insignificant rise in obese subjects as compared to non-obese subjects.

We found that the systolic blood pressure response (mmHg) to cold pressor test showed less rise in obese subjects ( $7.77 \pm 3.19$ ) as compared to non-obese subjects ( $10.9 \pm 3.73$ ), which was statistically highly significant ( $p < 0.0001$ ).

Our findings were consistent with the findings of Grewal S et al (2011),<sup>[6]</sup> Garg R et al (2013),<sup>[19]</sup>

In our study, the diastolic blood pressure response to cold pressor test showed more rise in obese subjects ( $6.73 \pm 2.15$ ) as compared to non-obese subjects ( $6.47 \pm 2.45$ ), which was statistically insignificant ( $p > 0.05$ ). Our findings were consistent with the findings of Grewal S et al (2011),<sup>[6]</sup>

## **CONCLUSION**

Obesity is associated with both sympathetic and parasympathetic nervous system dysfunctions 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 are prone to weight gain and are at risk of various cardiovascular complications resulting from autonomic function. Obesity should be controlled by Regular exercise, Pranayama and Yoga. As there has been limited research work done on autonomic function tests in obese subjects, further more research is recommended.

## **REFERENCES**

1. Esler M, Straznicki N, Eikelis N, Masuo K, Lambert G, Lambert E. Mechanisms of sympathetic activation in obesity-related hypertension. 2006;48:787-96.
2. Ganong WF. Review of Medical Physiology. 22nd ed USA: Mc Graw- Hill Company; 2005.
3. Global health indicators part III. Risk factors. World Health Statistics. 2012;p112.
4. Guyton AC, Hall JE. Text book of medical physiology. 11th ed Singapore: W.B.Saunders; 2006.
5. Steering Committee. The Asia-Pacific perspective: Redefining obesity and its treatment. Melbourne: International Diabetes Institute. 2000.
6. Grewal S, Gupta V. Effect of obesity on autonomic nervous system. Int J Cur Bio Med Sci 2011;1(2):15-8.
7. Colak R, Donder E, Karaoglu A, Ayhan O, Yalniz A. Obesity and the activity of autonomic nervous system. Turk J Med Sci. 2000; 30: 173-176.
8. Davy KP, Orr JS. Sympathetic nervous system behavior in human obesity. Neuroscience and Biobehavioral Reviews 33 (2009) 116–124
9. 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 Dec;261(6 Pt 2):R1418-23.
10. Hofmann KL, Mussgay L, and Ruddle H. Autonomic cardiovascular regulation in obesity. J Endocrinol 2000;164: 59-66.
11. Peterson HR, Rothschild M, Weingberg CR, Fell RD, Meleish KR, Pfeifer MA. Body fat and the activity of autonomic nervous system. N Eng J Med 1988;28:1077-83.
12. Arone LJ, Mackintosh R, Rosenbaum M, Leibel RL, Hirsh J. Autonomic nervous system activity in weight gain and weight loss. Am J Physiol 1995 Jul;269(1pt2):R222-5.
13. Rossi M, Marti G, Ricordi L, Fornasari G, Finardi G, Fratino P et al. Cardiac autonomic dysfunction in obese subjects. ClinSci 1989;76:567-72.
14. Jain AK. Manual of practical Physiology. 3rd ed Himachal Pradesh: Arya publications; 2008.
15. Mahajan BK. Methods in biostatistics. 7th ed Jaypee brothers publications; 2010.
16. Plaengdee J, Khrisanpant W, Boonsawat W, Pasurivong O, Zaeoue U. Effects of obesity on cardiac autonomic activity in adult. 12th National graduate conference. 2009 Feb.
17. Alvarez GE, Beske SD, Ballard TP, Davy KP. Sympathetic neural activation in visceral obesity. Circulation. 2002;106:2533-6.
18. Garg R, Malhotra V, Goel N, Dhar U, Tripathi Y, Medical S. A study of autonomic function tests in obese people. International Journal of Medical Research. 2013;2(4):750-5.
19. Grassi G, Seravalle G, Cattaneo BM, Bolla GM, Lanfranchi A, Colombo M et al. Sympathetic activation in obese normotensive subjects. 1994 Nov 11.
20. Sarwari KN. Assessment of autonomic (sympathetic and parasympathetic) cardiovascular and respiratory parameters in young obese males and females in the age group 18-25 years. Dissertation, Rajiv Gandhi university of health sciences. 2011.