Effect of obesity on parasympathetic nervous system

Supriya Bajirao Sakhare¹, Ajit Ashok Holkunde^{2,*}

Assistant Professor, ¹Dept. of Physiology, ²Dept. of Anatomy, Smt. Kashibai Navale Medical College, Pune, Maharashtra, India

*Corresponding Author:

Email: drholkunde@gmail.com

Received: 9th January, 2018	Accepted: 12 th February, 018
,,,,	j,

Abstract

In the present study, obese subjects (body Mass Index $>30 \text{ kg/m}^2$) and non-obese subjects (Body Mass Index $<25 \text{ kg/m}^2$) were selected with to study the effect of obesity on the parasympathetic nervous system. To test the functioning of parasympathetic nervous system in these two groups, heart rate variation during deep expiration and during deep inspiration (E/I ratio) as well as heart rate variation during supine and immediately in standing position (30:15 ratio) were calculated. The expiration/inspiration ratio was significantly lower in obese subjects, as compared to non-obese subjects. Thus, obesity is associated with altered parasympathetic nervous system which may result in various cardiovascular complications.

Keywords: Body Mass Index, Expiration/inspiration ratio, Parasympathetic nervous system.

Introduction

Obesity which was more prevalent in developed countries is now progressively increasing in the developing countries also.¹ This has been called "New world syndrome". Obese people have greater risk of developing both cardiovascular and metabolic diseases like hypertension, atherosclerosis, diabetes mellitus and gallbladder diseases.² Obesity is a condition of excess fat deposition in the body.³

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.⁴ Increase in body weight is usually associated with increase in mean heart rate.⁵

As the autonomic nervous system plays important role in energy metabolism, obesity may have an alteration in their autonomic nervous systems.

Hence, in the present research work, we have studied functioning of parasympathetic nervous system in obese subjects.

Material and Methods

The present study is a case- control study which is a type of observational study. The sample size is calculated with the graph pad prism 5 software. In the present study, total 120 subjects were selected from general population. Number of subjects in case group (obese) were 60 and number of subjects in control group (non-obese) were 60. They all belonged to middle socio-economic class.

Selection criteria

Study group: Age group: 25-50 years. Subjects had Body Mass Index >30 kg/m², of class I & class II. In case of female subjects, they were in follicular phase of their regular menstrual cycle.

Control group: Age group: 25-50 years. Subjects had Body Mass Index $<25 \text{ kg/m}^2$. In case of female subjects, they were in follicular phase of their regular menstrual cycle.

Exclusion criteria

- 1. Subjects of hypertension and other cardiovascular disorders, diabetes, renal, hepatic and any other disorders.
- 2. Subjects with history of smoking/alcohol/drug abuse.

After explaining the procedures of tests to subjects, the physical examination of all the subjects was done. The consent form was signed by the subjects.

Methodology

Parasympathetic function tests: The parameter of these tests was heart rate (beats/minute).

- 1. Heart rate (beats/minute): A continuous ECG (only lead II) of subject was recorded for 1 minute. Heart rate (per minute) =1500/R-R interval in mm⁶
- 2. Heart rate was measured during expiration and during inspiration in deep breathing (6 breaths per minute) to calculate the ratio. (E/I ratio) A value of 1.20 or higher was taken as normal.
- **3.** Heart rate response was measured immediately when subject stands from supine position. (30:15ratio). Longest R-R interval at beat 30 and shortest R-R interval at beat 15 after standing was measured to calculate ratio. Value of <1.00 was considered as abnormal.

All the data was presented as mean \pm S.D. (Standard Deviation) and statistical analysis was done with the student's unpaired 't' test.

Results

The mean body mass index of study group was 32.75 ± 2.02 and of control group was 22.44 ± 1.78 . This difference in mean body mass was statistically highly significant (p<0.0001).

The resting heart rate was insignificantly higher (p>0.05) in obese subjects (74.93 \pm 8.33) as compared to non-obese subjects (74.57 \pm 9.07).

Expiration/inspiration ratio was lower in obese subjects (1.13 ± 0.12) as compared to non-obese subjects (1.25 ± 0.08) , which is statistically highly significant (p<0.0001).

30:15 ratio is insignificantly (p>0.05) decreased in obese subjects (1.05 ± 0.12) as compared to non-obese subjects (1.06 ± 0.05) .

 Table 1: Comparison of parameters in obese and non-obese subjects

	Obese subjects	Non-obese subjects	t value	p value
BMI	32.75 ±	22.44 ±	29.47	< 0.0001*
(kg/m^2)	2.02	1.78		
HR	$74.93 \pm$	$74.57 \pm$	0.23	0.82 NS
	8.33	9.07		
30:15	$1.05 \pm$	1.06 ± 0.05	0.41	0.68 NS
ratio	0.12			
E/I ratio	1.13 ±	1.25 ± 0.08	6.42	< 0.0001*
	0.12			

NS = Non Significant, * = Significant

Discussion

In the present research, it was found that the mean body mass index of obese subjects was 32.75 ± 2.02 kg/m² and that of non-obese subjects was 22.44 ± 1.78 kg/m². The BMI difference between obese and non-obese subjects was statistically highly significant (p<0.0001).

In the present study, similar to other workers, the resting heart rate (beats/minute) was insignificantly (p>0.05) higher in obese subjects. Expiration/inspiration ratio showed statistically highly significant (p<0.0001) decrease in obese subjects as compared to non-obese subjects. Similar results have been reported by other workers.^{5,7-9}

Our study showed that 30:15 ratio was lower in obese than non-obese subjects which is statistically insignificant (p>0.05).

Our finding matches with findings of Colak R *et al* (2000),⁴ Grewal S *et al* (2011)¹⁰ and Thorat K *et al* (2011).⁹

Rossi M *et al* (1989),¹¹ Akhter S *et al* (2011),¹² Garg R *et al* (2013)¹³ found that 30:15 ratio was significantly lower in obese subjects as compared to non-obese subjects.

Cardiac parasympathetic dysfunction in obese subjects might be due to following reasons:

- 1. The neural control of the heart is regulated through the interplay of the sympathetic and vagal outflows. In most physiological conditions, the efferent sympathetic and parasympathetic branches have opposing actions: the sympathetic system influencing automaticity, whereas the parasympathetic system inhibits it. Studies have demonstrated either a reduced or unaltered parasympathetic activity in obese adults compared to lean subjects.¹⁴
- 2. Increase in body weight above the usual or starting weight, was associated with rise in mean heart rate (beats/min).⁵
- 3. Cardiac dysfunction in obese subjects may be due to excess intake of carbohydrate than fat and protein. It is seen that, there is increase in the parasympathetic activity with weight loss in obese subjects.¹⁰
- 4. Peterson HR *et al*¹⁵ suggested a correlation between increasing percentages of body fat and depression in parasympathetic activity. Whereas Garg R *et al* $(2013)^{13}$ indicated decrease in parasympathetic nerve function and baroreflex sensitivity in obese subjects.⁹ This baroreceptors resetting may be due to atherosclerosis that hardens the carotid sinus walls which decreases compliance.¹³
- 5. In obese subjects, parasympathetic damage or decreased vagal tone may occur due to hyperinsulinemia or insulin resistance or there may be decrease in baroreflex activity.¹²
- 6. In obese people, inhibition of lipid oxidation rate is more which could be due to decrease in parasympathetic activity.¹⁶
- 7. In obesity, number of insulin receptors per cell decreases. Adipose tissues in obese persons are more resistant to insulin actions than normal adipose tissue. There is hyperinsulinemia and dyslipidemia in association with obesity. This is called as the metabolic syndrome or syndrome X.¹⁷
- Obesity is always associated with increase in 8 plasma triglycerides. Dyslipidemia may also cause dysfunction. Total autonomic cholesterol, triglyceride, and low-density lipoprotein were negatively correlated with parasympathetic function tests; whereas high-density lipoprotein was positively correlated with parasympathetic function. Hypercholesterolemia has been proved to be associated with decreased 24 hours heart rate variability in men with or without coronary heart disease. Baroreceptor sensitivity is negatively correlated with low-density lipoprotein cholesterol levels. The impaired endothelium dependent arterial dilatation in vessel walls caused by higher lipid levels might also change the baroreflex capacity. Lower heart rate variability was proven to

Indian Journal of Clinical Anatomy and Physiology, April-June, 2018;5(2);154-156

be associated with a greater risk for developing hypertension among normotensive men and hypertension is one of the major risk factor of coronary heart disease.¹⁸

Summary and Conclusions

In study of parasympathetic function tests in obese subjects, statistical analysis showed:

- 1. The resting heart rate (beats/minute) was insignificantly higher in obese subjects as compared to non-obese subjects.
- 2. Statistically significant decrease in values of heart rate during deepnbreathing (E/I ratio) but heart rate response to immediate standing (30:15 ratio) was insignificantly lower in obese subjects as compared to non-obese subjects.
- 3. In obesity, dysfunction in parasympathetic nervous system is seen. Early diagnosis of such dysfunction will be helpful to reduce the risk and to avoid complications of cardiovascular system as well.
- 4. Obesity should be controlled by Regular exercise, Pranayama and Yoga.
- 5. As there has been limited research work done on autonomic function tests in obese subjects, further more research is recommended.

Limitation of study

The potential bias of present study is the selection of obese group. The exclusion criteria used is subjects of hypertension, liver and kidney diseases. Though, the subjects of obese group are not having any such diseases, pathogenesis of all these diseases may have arisen which may alter the result.

References

- 1. Esle M, Straznicky 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. Steering Committee. The Asia-Pacific perspective: Redefining obesity and its treatment. Melbourne: International Diabetes Institute. 2000.
- Colak R, Donder E, Karaoglu A, Ayhan O, Yalniz A. Obesity and the activity of autonomic nervous system. Turk J Med Sci. 2000;30:173-6.
- 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.
- Srinath C.G, Sarath R. Cardiovascular responses to isometric handgrip exercise test in obese and normal weight medical students. Dissertation, Rajiv Gandhi University of health sciences. 2010 May 16:1-73.
- Grassi G, Seravalle G, Cattaneo BM, Bolla GM, Lanfranchi A, Colombo M *et al.* Sympathetic activation in obese normotensive subjects. 1994 Nov 11.
- 8. Matsumoto T, Miyawaki T, Ue H, Kanda T, Zenji C, Moritani T. Autonomic responsiveness to acute cold

exposure in obese and non-obese young women. International Journal of Obesity 1999;23:793-800.

- Thorat KD, Ghuge SH. A prospective observational study of the correlation between obesity and the autonomic nervous system. Clinical research. International Journal of Biomedicine 2011;1(3):128-31.
- Grewal S, Gupta V. Effect of obesity on autonomic nervous system. Int J Cur Bio Med Sci 2011;1(2):15-8
- Rossi M, Marti G, Ricordi L, Fornasari G, Finardi G, Fratino P *et al.* Cardiac autonomic dysfunction in obese subjects. Clin Sci 1989;76:567-72.
- Akhter S, Begum N, Ferdousi S. Autonomic Neuropathy in Obesity. J Bangladesh Soc Physiol 2011 June; 6(1):5-9.
- 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.
- Plaengdee J, Krisanpant W, Boonsawat W, Pasurivong O, Zaeoue U. Effects of obesity on cardiac autonomic activity in adult. 12th National graduate conference. 2009 Feb.
- 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.
- 16. Valensi P, Lormeau B, Dabbech M, Miossec P, Paries J, Dauchy F *et al.* Glucose induced thermogenesis, inhibition of lipid oxidation rate and autonomic dysfunction in non-diabetic obese women. International Journal of Obesity. 1998;22:494-9.
- 17. Jain AK. Textbook of physiology. Volume-I. 5thed New Delhi: Avichal publishing company;2012.
- Chaudhuri A, Borade NG, Saha S. Relationship of gender and lipid profile with cardiac parasympathetic reactivity. Journal of basic and clinical reproductive sciences 2012 Jan-Dec;Vol 1(1,1):30-3.

Abbreviations: BMI - Body Mass Index, HR - Heart rate.