

## BODY MASS INDEX (BMI) BASED STUDY OF HEART RATE VARIABILITY IN YOUNG ADULTS

Neeru Garg\*, Priyanka Gupta\*\*, Punam Verma\*\*\*, Nidhi Jain\*\*\*, Sunita Mittal\*\*\*\*, Satendri Devi\*\*\*\*\*

\*Associate Professor\*, MD student\*\*, Professor\*\*\*, Assistant Professor\*\*\*\*, Department of Physiology, SGRRIM&HS, Dehradun-248001.  
Associate professor \*\*\*\*Department of Physiology, AIIIMS Rishikesh

**Abstract : Background & Objectives :** HRV is one of the most promising and popular markers of cardiac autonomic activity. It is a non invasive technique having significant diagnostic, clinical and research application. Rapid weight gain is associated with increased cardiac sympathetic tone in humans. There is a lack of autonomic imbalance studies involving young obese individuals. This study is an effort to assess the effect of obesity on cardiac autonomic activity using Heart Rate Variability in young adults in normal weight and obese subjects. **Methods:** 140 young adults 18-25 years were categorized into BMI based, normal and obese groups. Raw data for HRV analysis was a 'lab chart recording' of heartbeat signal from a pulse transducer in a resting subject for 5 min. at 1000 samples/sec. **Results:** There was a significant ( $p < 0.01$ ) decrease in SDNN, E/I ratio, HF nu and a significant ( $p < 0.01$ ) increase in LF nu, LF/HF in cases when compared to controls. **Interpretation & Conclusion:** In Obese group, study showed a statistically significant reduction in parasympathetic activity and a statistically significant increase in sympathetic activity. There was a shift in the sympathovagal balance towards sympathetic predominance among obese young adults in contrast to normal young adults.

**Key words:** HRV, body mass index, obesity, autonomic imbalance.

**Author for correspondence** – Dr. Neeru Garg, Associate Professor, 30, Subhash Road, Lane opposite Police Head quarters, Dehradun - 248001. E mail: [drneeruag@gmail.com](mailto:drneeruag@gmail.com)

### Introduction:

There is a lack of autonomic imbalance studies involving young obese individuals. Nutritional problem in India is gradually shifting from undernourishment to obesity<sup>1</sup>. It is a condition, which has evolved with the advent of civilization, sedentary life style and high calorie diet<sup>2</sup>. Obesity is one of the causative factors for multiple co-morbid conditions leading to metabolic and cardiac disorders<sup>3</sup>. Growing number of evidences indicate association of obesity and sudden cardiac deaths<sup>4,5</sup>. Heart Rate Variability (HRV) is a specific and sensitive non invasive tool to evaluate cardiac autonomic activity. HRV is the degree of variation of the heart rate under the balanced influence of sympathetic and parasympathetic components of the cardiac autonomic nervous system. HRV also indicates the extent of neuronal damage to autonomic nervous system<sup>7</sup>.

HRV represents continuous fluctuations in heart rate. R-R interval variations in ECG represent beat to beat control of heart rate mainly by the autonomic nerve supply to heart. Sympathetic and parasympathetic activities directed to the sinus node characterized by each cardiac cycle can be modulated by central and peripheral stimulators. These stimulations generate rhythmic fluctuations in efferent neural discharge that manifest as oscillations in the heart beat period. HRV denotes the variability's of both instantaneous HR and consecutive RR intervals. Cardiac autonomic function can be quantified by short term and long term HRV analysis<sup>8</sup>. Thus HRV measured by power spectral analysis provides a quantitative marker of autonomic neural control of heart rate and has been shown to reflect cardiovascular health. Low HRV is associated with an increased risk of coronary heart disease and sudden cardiac death.<sup>9,10</sup>

Time domain analysis of HRV uses statistical methods to quantify the variation of the standard deviation or the differences between

successive R-R intervals. Frequency domain analysis of HRV enables us to calculate the

respiratory dependent High Frequency (HF) and the Low Frequency (LF) powers<sup>11</sup>.

High frequency power is mediated by vagal activity, while low frequency power has been suggested to represent both sympathetic and parasympathetic activity but predominantly sympathetic modulation. Whereas LF/HF ratio mirror sympathovagal balance or reflect the sympathetic modulation<sup>12,13,14</sup>.

HRV studies among adults and children with obesity have revealed inconsistent results including high and low sympathetic tones coupled with a reduction in vagal tone<sup>15,16,17</sup>. It has also been shown in western studies on obese adults that weight loss reverses back to parasympathetic control of cardiac functions.<sup>18</sup>

While there is evidence of significant changes in autonomic control of cardiac functions in obese children and adults, there is lack of information concerning changes in obese young adults and also studies regarding HRV in obese Indian population are very few. Hence present study was undertaken with the aim of evaluating the resting cardiac autonomic nerve activity given by the changes in HRV analysis in healthy obese young adults.

This study is an effort to assess the effect of obesity on cardiac autonomic activity using Heart Rate Variability in young adults in normal weight and obese subjects.

### Materials & Method:

Study was carried out in Department of Physiology, SGRRIM&HS, Dehradun after permission from Institutional Ethics Committee.

**Subjects chosen :** 140 young adults 18-25 years after seeking their informed consent. Anthropometric parameters like height and weight were recorded. Body Mass Index (BMI) was calculated by WHO criterion<sup>19,20</sup>. Out of 140 participants ,100 subjects who fulfilled the selection criterion were selected. Overweight subjects were excluded.

They were categorized as Cases: Obese (n=50) and Controls :Normal weight (n=50) based on the BMI. Controls whose BMI < 25kg/m<sup>2</sup> (18.5-24.9) and Cases with BMI > 30kg/m<sup>2</sup>. Subject's clinical history and details were taken . Diseases like diabetes mellitus, hypertension, endocrinal disorders and any history of drugs intake affecting the Autonomic Nervous System like adrenergic blockers, calcium channel blockers, others were also excluded.

Raw data for HRV analysis was obtained from a 'lab chart recording' of heartbeat signal from a pulse transducer in a resting subject for 5 min. at 1000 samples/sec with LAB CHART PRO-7. Results were shown in a point care plot , period histogram, delta RR histogram, tachogram , spectrum and statistical report (fig-1).

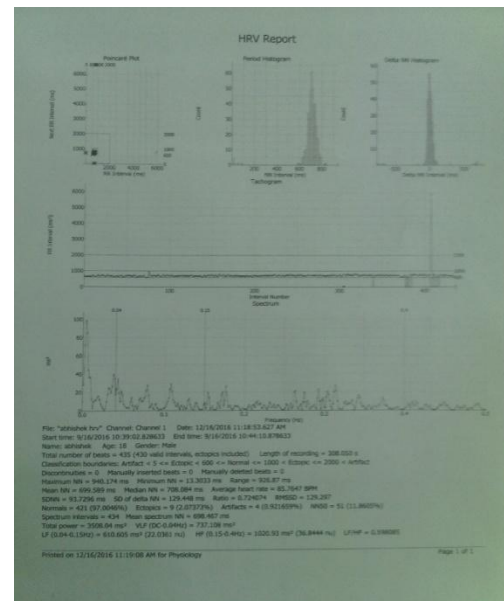


Fig 1

### Statistical analysis:

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented as Mean  $\pm$  sd. Significance is assessed at P value <0.05 level of significance. Student 't' test (two tailed , unpaired) has been used to find the significance

of study parameters on continuous scale between two groups, 'Inter group analysis').

#### Observation & Results:

**TABLE 1: Comparison of basic characteristics between control & cases**

Variables	Controls	Cases	P value
Age (years)	20.98± 2.38	20.62± 2.41	0.45
BMI (kg/m <sup>2</sup> )	21.39± 2.15	31.77± 2.24	<0.05*

The mean age of the controls was 20.98± 2.38 and of cases was 20.62± 2.41 respectively.

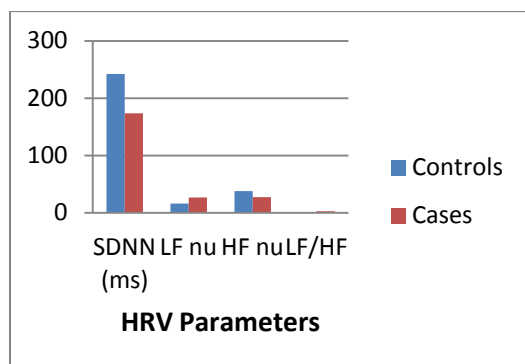
The BMI mean ± sd of controls was 21.39 ± 2.15 and of cases was 31.77 ± 2.24 which was significantly higher in cases (p<0.05).

Table-2 shows significantly ↓ SDNN, ↓HF component in cases when compared to controls suggesting the reduction in the parasympathetic activity.

Significant ↑ LF component and ↑LF/HF ratio shows elevated cardiac sympathetic activity in obese individuals.

Thus, the study results showed significant relationship between BMI variation and HRV parameters.

**Figure-2 : Comparison of time domain and frequency domain parameters between controls & cases**



**TABLE 2: Comparison of Time Domain and Frequency Domain parameters.**

HRV	Controls	Cases	P value
SDNN (ms)	242.1±125.1 (79.8-366.2)	173.7±124.8 (86.0-444.0)	0.007**
LF nu	16.1 ± 10.2 (7.1 – 62.2)	26.5 ± 16.7 (3.5 – 37.2)	0.002**
HF nu	37.87 ± 17.9 (7.4 – 59.8)	27.4 ± 18.2 (6.1 – 65.0)	0.009**
LF/HF	0.6±0.3 (0.27- 1.1)	2.47 ± 0.23 (0.29-4.7)	0.01*

Values : Mean ± sd

P value < 0.05 – Significant

< 0.001- Highly significant

#### Discussion:

The major findings of this study shown in (table-2 ,fig-2) are (↓SDNN, ↓E/I ratio, and ↓HF component in cases as compared to controls) indicates the presence of impaired parasympathetic activity and (significant ↑ LF component and ↑LF/HF ratio) shows elevated level of sympathetic activity in obese cases.

Previous studies<sup>21,22,23,24</sup> showed a significant reduction in the parasympathetic activity with increasing body weight .They gave explanation similar to present study that , ANS dysfunction of the obese appears to be the direct result of day long hyperinsulinemia, due to low energy expenditure and low glucose and fat metabolism and it is relevant that insulin is able to directly affect cell excitability (through its action on transmembrane ion exchange) and permeate the bloodbrainbarrier to modulate neuronal activities in the midbrain.

Increase in sympathetic activity was observed in obese in present study, but this is in contrast to other studies, which reported that, if the obesity is

of a longer duration, then it is likely to lead to global reduction of the autonomic activity and hence a reduction in the sympathetic activity also<sup>25</sup>.

HF in power is the direct representation of vagal tone. In the present study HF in power and HF nu units are significantly decreased in the obese group compared to control group, indicating early cardiovascular vagal tone changes in the obese. Vagal tone is an important determinant of cardiovascular health. Vagal tone of an individual has insightful influence on the heart rate, cardiac output and blood pressure. Persons with poor vagal tone are more prone to develop cardiovascular diseases such as myocardial infarction, hypertension and heart failure. A lower SDNN in present study indicates diminished baroreceptor reflex modulation of RR intervals. Low SDNN and low HF power, taken together, which are seen in the study group is indicative of poor vagal control in the cardiovascular system<sup>26</sup>.

Sympathovagal imbalance explains increased incidence of sudden cardiac deaths associated with obesity. Thus early interventional programs like **weight reduction, life style changes and physical exercises**, which reduce fat content of the individual, can be advised to reduce the chances of subsequent cardiac abnormalities.

#### Summary and Conclusions:

Present study showed altered cardiac autonomic activity in obese individuals. Obese group showed statistically significant reduction of parasympathetic activity and statistically significant increase in sympathetic activity in obese subjects.

The duration of obesity was not considered in this study, which could have helped in establishing the relation of duration & effects of obesity on cardiac autonomic activity.

There is scope for further studies: it could be undertaken in various age groups and also in both genders for more clarity. Further a prospective study can be undertaken in the same subjects to know the effect of weight loss on the cardiac autonomic activity.

#### References:

1. Nageswari SK, Sharma R, Kohli DR. Assessment of respiratory and sympathetic cardiovascular parameters in obese school children. *Indian J Physiol Pharmacol* 2007; 51(3):235-43.
2. Younghee K, Youn KS, Haymie C. MBI and metabolic disorders in South Korean adults: Korea National Health and Nutrition Survey. *Obes Res* 2004;12: 445-453.
3. Yuko A, Hironobu Y, Mamoru K, Toshie S, Hiroshi E, Sukenobu I. VLCD Induced weight loss improves heart rate variability in moderately obese Japanese. *Exp Biol Med* 2001 ;226(5):440-5.
4. Kirsten LR, Harry H, Meena K, Eric B, Marek M, Michael M. Effects of moderate and vigorous physical activity on heart rate variability in british study of civil servants. *Am J Epidemiol* 2003;158:135-43.
5. Frenco R, Bernard S, Andrea C, Tiziana G, Barbara DV, Ivana R et al. Assessment of cardiac autonomic modulation during adolescent obesity. *Obes Res* 2003 April;11(4):541-548.
6. Jeong AK, Young GP, Kyung HC, Myung HH, Hee CH, Youn SC, Dokyung Y. Heart rate variability and obesity indices: Emphasis on the response to noise and standing. *J Am Board Fam Pract* 2005;18:97-103.
7. Task force of the European society of cardiology and the North American society of pacing and electrophysiology. Heart rate variability: standards of measurement, physiological interpretation and clinical use. *European Heart Journal* 1996; 17: 354-81.
8. Muralikrishnan K, Balasubramanian K, Jawahar ali S. M, Rao B V. Poincare plot of heart rate variability: an approach towards explaining the cardiovascular autonomic function in obesity. *IJPP* 2013; 57(1) : 31-37.
9. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation and clinical use. *Circulation* 1996; 93: 1043- 65.
10. Liao D, Cai J, Rosamond D, Barnes R, Hutchinson R. Cardiac autonomic function and incident

- coronary heart disease—The ARIC study. *Circulation* 1995; 92(Suppl 1):418.
11. Moolgard H, Sorensen K, Bjerregaard P. Attenuated 24-h heart rate variability in apparently healthy subjects, subsequently suffering sudden cardiac death. *Clin Auton Res* 1991;1:233–7.
  12. Hayano J, Sakakibara Y, Yamada A, Yamada M, Mukai S, Fujinami T, et al. Accuracy of assessment of cardiac vagal tone by heart rate variability in normal subjects. *Am J Cardiol* 1991;67:199–204.
  13. Pomeranz B, Macaulay RJ, Caudill MA, Kutz I, Adam D, Gordon D, et al. Assessment of autonomic function in humans by heart rate spectral analysis. *Am J Physiol* 1985; 248: H151–3.
  14. Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. *Circulation* 1991;84:1482–92.
  15. Karason K, Molgaard H, Wikstrand J, Sjostrom L: Heart rate variability in obesity and the effect of weight loss. *Am J Cardiol* 1999;83:1242–7.
  16. Gao YY, Lovejoy JC, Sparti A, Bray GA, Keys LK, Partington C: Autonomic activity assessed by heart rate spectral analysis varies with fat distribution in obese women. *Obes Res* 1996;4:55–63.
  17. Piccirillo G, Vetta F, Viola E, Santagada E, Ronzoni S, Cacciafesta M, et al: Heart rate and blood pressure variability in obese normotensive subjects. *Int J Obes Relat Metab Disord* 1998;22:741–50.
  18. Akehi Y, Yoshimatsu H, Kurokawa M, Sakata T, Eto H, Ito S, et al. VLCD-Induced Weight Loss Improves Heart Rate Variability in Moderately Obese Japanese. *Exp Biol Med* 2001;226:440–5.
  19. William DM, Katch FI, Katch VL. Essentials of exercise physiology : Body composition, obesity and weight control. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2006. 558-627.
  20. Park K. Text book of preventive and social medicine: Epidemiology of Non-Communicable Disease: Obesity. 19th ed. Jabalpur: Banarsidas publishers Bhanot; 2007. 332-6.
  21. Emdin M, Gastaldelli A, Muscelli E. Hyperinsulinemia and autonomic nervous system dysfunction in obesity: effects of weight loss. *Circulation* 2001; 103:513–9.
  22. Martini G, Riva P, Rabbia F, Molini V, Ferrero GB, Cerutti F et al. Heart rate variability in childhood obesity. *Clin Auton Res* 2001 Apr; 11(2):87-91.
  23. Facchini M, Malfatto G, Sala L, Silvestri G, Fontana P, Lafortuna C et al. changes of autonomic cardiac profile after a 3-week integrated body weight reduction program in severely obese patients. *J Endocrinol Invest* 2003 Feb;26(2):138-42.
  24. Poirier P, Hernandez TL, Weil KM, Shepard TJ, Eckel RH. Impact of diet induced weight loss on the cardiac autonomic nervous system in severe obesity. *Obes Res* 2003 Sep; 11(9):1040-7.
  25. Masuo K, Mikami H, Ogihara T, Tuck ML. Weight gain-induced blood pressure elevation, Hypertension 2000;35:1135-40.
  26. Muralikrishnan K, Balasubramanian K, Rao B V. Heart rate variability in normotensive subjects with family history of hypertension. *IJPP* 2011; 55 (3):253–561.

**Disclosure:** No conflicts of interest, financial, or otherwise are declared by authors