



Research Journal of Pharmaceutical, Biological and Chemical Sciences

A Comparative Study of Heart Rate Variability in Obese and Healthy Young Adults (18 – 25 Years).

Ravi Kant Soni*, Jyotsna Shukla, Amitabh Dube, Arvind Kumar Shukla, Ranjeeta Soni,
Shashi Kant Soni.

ABSTRACT

The heart rate variability (HRV) represents fluctuations in the heart rate under the influence of autonomic nervous system. The assessment of heart rate variability proves to be the most sensitive, specific and valuable tool, which is a noninvasive index reflecting the sympathetic and parasympathetic components of the autonomic nervous system on the sinus rhythm. Our aim is to assess and compare the Heart Rate Variability in obese and (age and sex matched) healthy young adults (18–25 years). The Study group comprised of 35 obese young adults males in the age group of 18-25 years and confounded age and sex matched control group comprised 35 healthy young adults. Evaluation was done by recording the 5 min resting ECG in supine position. The analogue signal was converted to digital signal by using National Instrument software version 8.0 and HRV analysis was done with HRV software version 1.1. The significance of difference in the means of both group was inferred by unpaired 't' test. The Findings of this study reveals that total power was reduced, LF n.u. and LF/ HF ratio is significantly higher in obese group as compared to healthy control group (P 0.000). The study concluded that there is deranged cardiac autonomic function.

Key word: Heart Rate Variability, Body Mass Index, Autonomic Nervous System, Low Frequency, High Frequency.

**Corresponding author*

INTRODUCTION

Obesity can be seen as the first wave of a defined cluster of non-communicable diseases called “New World Syndrome,” creating an enormous socioeconomic and public health burden in developed economies [1]. Nutritional problem in India is also gradually shifting from undernourishment to obesity [2]. It is on rise in our society due to socioeconomic developments leading to change in lifestyle particularly dietary pattern [3, 4].

Approximately 60% of individuals with obesity have the metabolic syndrome (including three or more of the following factors: elevated abdominal circumference, blood pressure, blood triglycerides, fasting blood sugar, and low high-density lipoprotein [HDL] cholesterol [5]. It is associated with an increased risk of death from all causes compared to normal-weight individuals, mostly due to cardiovascular causes through various mechanisms [6, 7, 8].

It is well known that autonomic response is the first human response to any intervention or to any physical, physiological, pathological or psycho emotional activity [9]. There is information regarding the presence of autonomic disturbances (both sympathetic and parasympathetic) in obesity leading to an increased incidence of sudden cardiac death [10, 11].

Cyclic variability of sinus node cycle mainly depends on the dynamic oscillations in the activity of the sympathetic and parasympathetic autonomic nervous system. Among the different available non-invasive techniques for assessing the autonomic status, Heart Rate Variability (HRV) is a simple method to evaluate the sympathovagal balance at the sinoatrial level [12]. HRV also indicates the extent of neuronal damage to autonomic nervous system [13]. In a damaged heart, the changes in activity in the afferent and efferent fibers of the ANS and in the local neural regulation will contribute to the resulting sympathovagal imbalance reflected by a diminished HRV [11].

Spectral analysis of heart rate has proven to be a useful means to gain insight into sympathetic and parasympathetic control of heart activity [14]. It has also been shown in western studies on obese adults that weight loss reverses back to parasympathetic control of cardiac functions [15, 16].

Hence present study was under taken with the aim of evaluating the resting cardiac autonomic nervous system activity given by the changes in HRV in healthy obese young adults, as early establishment of this correlation will help in preventing future cardiac dysfunction.

MATERIAL AND METHOD

The present study was conducted in the Upgraded Department of Physiology, S.M.S. Medical College, Jaipur. The study was approved by Institutional Ethical committee. 35 obese (BMI 30.78 ± 0.78) young adult males of 18 – 25 years and 35 age matched healthy (BMI 20.54 ± 1.24) controls volunteered to participate in the study were randomly selected from Obesity Clinic, S.M.S. Medical College and Attached Hospitals.

Prior to recording, a detailed history and physical examination was done to exclude the Syndromic obesity, endocrinological disorders and other disease as Diabetes Mellitus, Hypertension or drugs interfering the autonomic nervous system. Coffee, nicotine or alcohol was avoided at least 24 hours prior to the testing and to avoid food 2 hours before testing. The examination was carried out between 10 AM to 1 PM at 24 – 250 c temperature.

The subjects were instructed to breathe quietly during the entire recording period with closed eyes and to avoid talking, moving hands, legs or body, coughing and sleeping. All standard limb leads and chest leads were applied and the lead with upright 'R' wave was selected. The assessment of Heart Rate Variability was done by recording the 5 minutes E.C.G in supine position by RMS ECG (DECG 1/ 63041/ ADBXB) after 15 min supine rest. The analogue signal was converted to digital signal by National Instrument software NI - DAQ Version 8.0. The analysis of Heart Rate Variability in Frequency domain measures was done by HRV Software Version 1.1. developed by AIIMS, New Delhi [17].

Heart rate Variability: In the Frequency Domain analysis the power spectrum for Heart Rate Variability was calculated with the fast fourier transformation (FFT) based method ¹³. Power spectral densities (PSD) were plotted in ms² / Hz against preset frequencies. Power of the spectral bands were calculated in ms² (absolute power) and in normalized units (n.u.). The power spectrum is subsequently divided into three frequency bands: very low frequency (VLF) range (0.001 – 0.04 Hz), Power in low frequency (LF) range (0.04 – 0.15 Hz) and Power in high frequency (HF) range (0.15 – 0.40 Hz).

Statistical Analysis: Numerical data are expressed as Mean ± S.D. The comparison of HRV indices between the two study groups (Obese and Healthy Young controls) was evaluated using unpaired student's 't' test. Statistical significance was assigned at p < 0.05.

RESULTS

The table 1 shows anthropometric characteristics of obese and healthy control subjects, highly difference was seen between two groups.

Table 1: Anthropometric characteristics of obese and healthy control subjects.

Parameters	Groups		p-value
	Obese (n=35)	Non obese (n=35)	
Height (Meters)	1.06 ± 0.03	1.68 ± 0.04	1.00
Weight (Kg)	86.91 ± 4.01	58.2 ± 4.23	0.000**
BMI (Kg / m ²)	30.78 ± 0.78	20.54 ± 1.24	0.000**

* Significant (p < 0.05), ** Highly significant (p < 0.01).

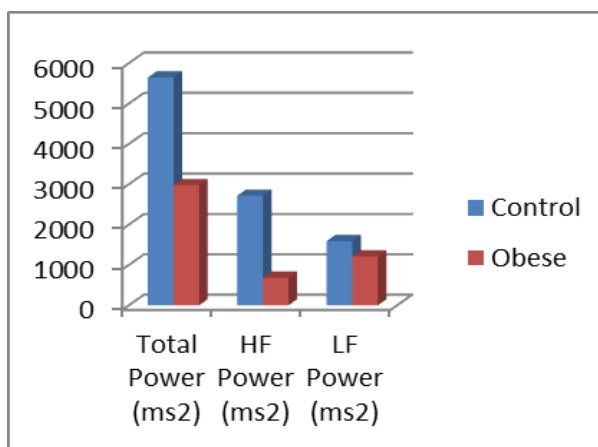
Total power & high frequency power in absolute terms are decreased in obese group as compared to healthy subjects. LF nu and LF/ HF ratio is increased and HF nu is decreased in obese as compared to healthy individuals results are statistically highly significant. Resting heart

rate is increased and low frequency power is obese but the results are statistically not significant (Table 2).

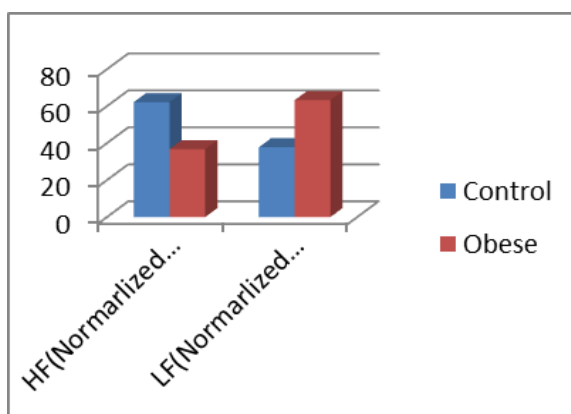
Table 2: Comparison of Means of various parameters of Heart Rate Variability between Obese young adults & Healthy control subjects.

Parameters	Groups		p-value
	Obese (n=35)	Non obese (n=35)	
Resting Heart Rate (bpm)	82.94 ± 10.90	80.66 ± 9.81	0.361
Total Power (ms ²)	2982.20 ± 1481.88	5648 ± 3275.32	0.000**
LF Power (ms ²)	1213.61 ± 747.31	1595.36 ± 1243.23	0.124
LF nu (%)	63.26 ± 11.89	37.78 ± 11.01	0.000**
HF Power (ms ²)	688.64 ± 436.82	2719.73 ± 1856.76	0.000**
HF nu (%)	36.74 ± 11.89	62.22 ± 11.01	0.000**
LF / HF Ratio	2.11 ± 1.52	0.66 ± 0.29	0.000**

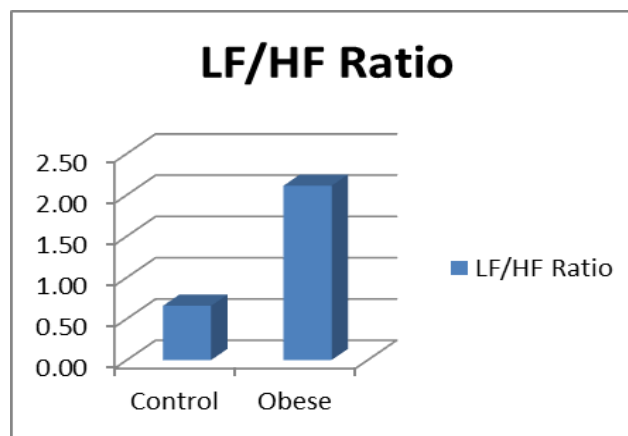
* Significant (p < 0.05), ** Highly significant (p < 0.01).



Histogram 1: comparison of Total Power, HF Power, LF Power between obese and healthy young adults.



Histogram 2: comparison of HF and LF normalized units between obese and healthy young adults.



Histogram 3: comparison of LF / HF ratio between obese and healthy young adults.

DISCUSSION

In the present study we concluded that all frequency domain parameters of HRV except LF nu and LF / HF ratio were reduced in obese study group. LF / HF ratio, a marker of sympathetic vagal balance and LF nu were increased in obese study group. Obese young adults had markedly depressed HRV, which is an expression of cardiac autonomic modulation and so it reflects cardiac autonomic disturbance in obese young adults. Depressed HRV, which reflects both sympathetic and parasympathetic activity predict increased risk for subsequent cardiac events in obese young adults.

The exact mechanism that may cause impairment of parasympathetic nerve function has not yet been clearly established. Obesity is said to be a state of impaired glucose tolerance, hyperinsulinemia and insulin resistance. Acute insulin administration has been shown to reduce high-frequency power, a measure of respiratory sinus arrhythmia, during euglycemic hyperinsulinemia in normal- weight and obese subjects [18,19]. Thus, hyperinsulinemia may contribute to low cardiac vagal activity [20].

The LF/HF ratio has been proposed to be an accurate measure of the overall sympathetic vagal balance of the autonomic nervous system in which higher values indicate a more sympathetically driven cardiovascular system [21]. Our findings indicate that high frequency modulations of heart rate variability are lower in obese study group than in control subjects while LF / HF ratio which indicates sympathetic vagal balance, is higher in obese study group compared with the control group. This further reflects a compromised state of parasympathetics in obese study group.

Earlier studies on sympathetic nerve activity in obese persons have produced conflicting results. Some studies have shown decrease [22,23,24] and some increase in sympathetic activity in obesity [23,25,26]. Measurements of plasma and urinary catecholamine concentrations as indices of sympathetic nervous system activity have ranged from low through

normal to high [22,27,28]. Sympathetic nerve activity in skeletal muscle was increased in obese subjects but skin sympathetic nerve activity was not significantly different [29].

Insulin and leptin levels are elevated in obesity. Thus increased insulin and leptin levels are thought to increase sympathetic nervous system activity [30,31,32].

The major findings of this study indicate the presence of impaired parasympathetic activity and elevated level of sympathetic activity in obese group. Obese persons may suffer from an increased mortality risk due to cardiovascular disorders related to either lowered parasympathetic or altered sympatho vagal balance. Early detection and management by weight reduction and regular exercise can reduce the risk, as these are shown to increase HRV^{15,16}. HRV analysis can detect changes even before clinical signs appear [13].

CONCLUSION

In the present study we concluded that all frequency domain parameters of HRV except LF nu and LF / HF ratio were reduced in obese study group. LF / HF ratio, a marker of sympatho vagal balance and LF nu were increased in obese study group. Obese young adults had markedly depressed HRV, which is an expression of cardiac autonomic modulation and so it reflects cardiac autonomic disturbance in obese young adults. Depressed HRV, which reflects both sympathetic and parasympathetic activity predict increased risk for subsequent cardiac events in obese young adults.

BIBLIOGRAPHY

- [1] Lau DC. *Can Med Assoc J.* 2007; 176: 1103-6.
- [2] Nageswari SK, Sharma R, Kohli DR. *Indian J Physiol Pharmacol.* 2007; 51(3): 235-43.
- [3] Chhatwal J, Verma M, Riar SK. *Asia Pac J Clin Nutr.* 2004; 13(3): 231-5.
- [4] Mohan B, Kumar N, Aslam N, Rangbulla A, Kumbkarni S, Sood N K. *Indian Heart J.* 2004; 56: 310-4.
- [5] Samuel K, Lora EB, George AB, Steven B, David BA, Xavier PS, Yuling H, Robert H. *Circulation.* 2004; 110: 2952-2967.
- [6] Zimmet P, Alberti K G, Kaufman. *Pediatr Diabetes.* 2007; 8: 229-306.
- [7] Keskin M, Kurtoglu S, Kendirci M, Atabek ME, Yazici C. *Pediatrics.* 2005; 115: e500-e503.
- [8] Sangun O, Dundar B, Kosker M, Pirgon O, Dundar N. *J Clin Res Pediatr Endocrinol.* 2011; 3: 70-76.
- [9] Sztazel J: Heart rate variability. *Swiss Med. Wkly.* 2004; 134: 514-22.
- [10] Kirsten LR, Harry H, Meena K, Eric B, Marek M, Michael M. *Am J Epidemiol.* 2003; 158: 135-43.
- [11] Frenco R, Bernard S, Andrea C, Tiziana G, Barbara D V, Ivana R. *Obes Res.* 2003 April; 11(4): 541-548.
- [12] Tsuji H, Venditti F J Jr, Manders E S, et al. *Circulation.* 1994; 90: 878-883.

- [13] Task force of European society of cardiology and the North American society of Pacing and Electrophysiology. *Circulation*. 1996; 93: 1043-65.
- [14] Berntson G G, Bigger J T, Ekberg D L, Grossman P, Kaufmann P G, Malik M, Nagaraja H N, Porges S W, Saul J P, Stone P H, VanMolen M W. *Psychophysiology*. 1997; 34: 623-648.
- [15] Akehi Y, Yoshimatsu H, Kurokawa M, Sakata T, Eto H, Ito S, Ono J. *Exp Biol Med (Maywood)* 2001 May; 226(5): 440-5.
- [16] Sandercock GR, Bromley PD, Brodie DA. *Med Sci Sports Exerc*. 2005; 37(3): 433-9.
- [17] Deepak KK, Chatterjee K, AIIMS annual report. 2006; 462 – 474.
- [18] Muscelli E, Emdin M, Natali A, Pratali L, Camastra S, Gastaldelli A. *J Clin Endocrinol Metab*. 1998; 83: 2084-90.
- [19] Van de Borne P, Hausberg M, Hoffman R P, Mark A L, Anderson E. *Am J Physiol*. 1999; 276: R178-R183.
- [20] Valensi P, Paries J, Lormeau B, Attia S, Attali J R. *Metabolism*. 2005; 54: 1290-6.
- [21] Malliani A, Pagani M, Furlan R, Guzzetti S, Lucini D, Montano N. *Circulation*. 1997; 96: 4143-5.
- [22] Piccirillo G, Vetta F, Viola E, Santagada E, Ronzoni S, Cacciafesta M, Marigliano V. *Int J Obes Relat Metab Disord*. 1998 Aug; 22(8): 741-50.
- [23] Colak R, Donder E, Karaoglu A, Ayhan O, Yalniz M. *Turk J Med Sci*. 2000; 30: 173-6.
- [24] Akhter S, Begum N, Ferdousi S, Begum S, Ali T. *J Bangladesh Soc Physiol*. 2010 June; 5(1): 34-39.
- [25] Gao YY, Lovejoy JC, Sparti A, Bray GA, Keys LK, Partington C. *Obes Res*. 1996; 4: 55-63.
- [26] Karason K, Mølgaard H, Wikstrand J, Sjöström L. *Am J Cardiol*. 1999 Apr 15; 83(8): 1242-7.
- [27] Young JB, Macdonald IA. Sympathoadrenal activity in human obesity: heterogeneity of findings since 1980. *Int J Obes Relat Metab Disord*. 1992; 16: 959-67.
- [28] Grassi G, Vailati S, Bertinieri G, Seravalle G, Stella M L, Dell'Oro R, Mancia G. *J Hypertens*. 1998; 16: 1635-9.
- [29] Anderson EA, Balou TW, Hoffman RP, Sinkey CA, Mark AL. *Hypertension*. 1992; 19: 621-7.
- [30] Gudbjörnsdottir S, Elam M, Sellgren J, Anderson E A. *J Hypertens*. 1996; 14: 91-7.
- [31] Guízar JM, Ahuatzin R, Amador N, Sánchez G, Romer G. *Indian Pediatrics*. 2005; 42: 464-9.
- [32] Sjoberg N, Brinkworth GD, Wycherley TP, Noakes M, Saint DA. *J Appl Physiol*. 2011; 110(4): 1060-4.