

Relationship between heart rate variability and anthropometric parameters in young Indian adults

Shilpa Khullar¹, Praveena Sinha^{2*}, Mitasha Singh³, Diksha⁴

¹Associate Professor, ²Tutor, ³Assistant Professor, ⁴MBBS Student, ^{1,2}Dept. of Physiology, ⁴Dept. of Community Medicine, ESIC Medical College and Hospital, Faridabad, Haryana, India

***Corresponding Author: Praveena Sinha**

Email: amrita.amma6543210@gmail.com

Received: 4th February, 2019

Accepted: 4th April, 2019

Abstract

Introduction: Heart Rate Variability is a common non – invasive technique with significant diagnostic, clinical and research application. Obesity is seen to be associated with autonomic dysfunction with increased sympathetic activity and decreased parasympathetic activity in young people predisposing them to increased cardiovascular mortality and morbidity. The aim of our study was to see how obesity effects cardiac autonomic activity in normal weight and obese individuals.

Materials and Methods: The study was conducted on 60 young individuals between 18-25 years divided into two groups of 30 each based on their BMI: Group I – Normal weight group with BMI – 18-22.9 Kg/ m² and Group II – Obese subjects with BMI ≥ 25 Kg/m². HRV was done in both the groups and was analyzed for time domain and frequency domain parameters. The data was analyzed by using students ‘t test’ and Chi-square test. Correlation was done between various anthropometric measures and HRV using Pearson’s correlation coefficient.

Results: With the exception of height and neck circumference, all the anthropometric parameters were significantly higher in Group II compared to Group I While analyzing HRV for frequency domain parameters, LF (nu) and LF/HF were significantly lower in Group I compare to Group II. Time domain parameters of SDNN, RMSSD and pNN50 were significantly higher in Group I.

Conclusion: Our study showed a statistically significant reduction in parasympathetic activity and an increase in sympathetic activity in the obese compared to controls.

Keywords: Heart rate variability, Cardiac autonomic activity, Anthropometric parameters, Autonomic modulation, BMI.

Introduction

Obesity is gradually becoming a major nutritional problem across India while undernourishment continues to pose problems in the lower socio-economic strata.¹ It is a condition, which has evolved with the advent of civilization, sedentary life style and high caloric diet. Epidemiological data shows that prevalence rates are increasing both in industrialized and developing countries especially in the adolescent population due to socioeconomic development leading to change in lifestyle.^{2,3} According to WHO, one billion people are overweight and 300 million are obese worldwide.⁴ According to the database of Overseas Development Institute (ODI), the situation in our country is quite alarming with one in three of the total 1.46 billion overweight or obese adults worldwide being an Indian.⁵

The autonomic nervous system plays an important role, in controlling and maintaining both physiological functions as well as pathological conditions of the body. An increase in the percentage of fat content in the body beyond a permissible upper limit increases the risk of predisposition to various diseases. Etiopathogenesis of various non-communicable diseases like diabetic neuropathy, myocardial infarction, congestive heart failure, arrhythmias and sudden cardiac death have been seen to be associated with increased sympathetic activity and a reduced vagal tone.⁶⁻⁸

Heart Rate Variability (HRV) is a commonly used non-invasive research tool to see the sympatho-vagal balance at the level of the SA node.⁹ R-R interval variation in the ECG represents beat to beat control of heart rate by the autonomic

nerve supply to heart. Thus, HRV measured by time and frequency domain analysis is a quantitative marker of autonomic neural control of heart rate which is quite accurate and reproducible. Time domain analysis of HRV is a quantitative method to study the variation of the standard deviation or the difference between successive R-R interval. HRV analyzed in terms of the frequency domain parameters tells us about the respiratory dependent high frequency (HF) and low frequency (LF) powers. Low frequency power tell us about sympathetic and parasympathetic activity mainly sympathetic modulation, while high frequency power is mediated by vagal activity. LF/HF ratio mirrors overall sympatho-vagal modulation of autonomic nervous system on cardiac activity.⁹⁻¹⁰

Some authors have reported that, if the obesity lasts for a longer duration, it leads to an overall reduction in the autonomic activity, particularly in the sympathetic nervous system.¹¹ The high prevalence of sympathovagal imbalance in HRV in obese individuals in comparison to normal subjects predisposing them to higher cardiovascular morbidity early in life has been demonstrated by a number of similar studies done earlier.^{12-20,4}

As the rate of obesity in India and its sequel are rising steadily due to adoption of the Western lifestyle, the adverse effects of obesity on the cardiovascular system assume immense importance. However, such studies regarding HRV in adult Indian population are very few till date. Hence the present study was undertaken with the aim of evaluating the resting cardiac autonomic activity given by the changes in HRV analysis in healthy normal and obese young adults.

Materials and Methods

Study Design

Cross sectional study

This study was carried out in the Department of Physiology of a Government Medical College located in Delhi/ NCR region over a period of two months between August and September 2018. It was conducted following approval of the protocol from ICMR under their STS programme for MBBS students. The study was done on young Indian adult population of both the sexes aged between 18-25 years who were selected from the staff and students of the college. The purpose as well as procedure of conducting the study was explained to all the subjects and written informed consent was taken from them. Institutional Ethical Clearance (IEC) was taken following approval of the project from ICMR.

60 voluntarily consenting subjects were selected and divided into two groups of 30 each based on their BMI:

Group I - Normal weight group with BMI=18–22.9 Kg/m²

Group II - Obese group with BMI \geq 25 Kg/m²

The revised BMI cut-off for Asians recommended by WHO were used to classify the subjects as normal weight and obese.²¹

Subject Selection

Inclusion Criteria

1. Healthy young Indian adults of either sex aged between 18-25 years
2. Those with a resting heart rate of 60-80 beats per minute

Exclusion Criteria

1. History of chronic cardiac or pulmonary disease – hypertension, arrhythmias, myocardial infarction
2. Endocrine disorders
3. Those with painful conditions like arthritis and injuries
4. Epileptics
5. Long term treatment with drugs: anti-depressants, β -blockers, anti-arrhythmic, AC inhibitors, thyroid stimulators
6. Psychiatric disorders (depression, manic depressive illness etc.)
7. Symptomatic diabetic autonomic neuropathy
8. History of alcohol / tobacco abuse
9. Subjects doing regular physical exercise
10. Trained athletes

Recording of Anthropometric Parameters

Weight: Recorded using an electronic weighing scale to the nearest 0.5Kg

Height: It was measured using a standard meter scale in the standing position without footwear to the nearest 0.5 cms

Neck Circumference (NC): Measured in the midway of the neck between mid-cervical spine and mid anterior neck within 1 mm with the subject standing upright.

Waist Circumference (WC): Measured as the minimum circumference between the costal margin and the iliac crest measured in the horizontal plane with the subject standing.

Hip Circumference (HC): It is the maximum circumference in the horizontal plane measured over the buttocks.

All these anthropometric parameters were used to calculate the Body Mass Index (BMI), waist/hip ratio (W/H ratio) and the visceral body fat.

Recording of Heart Rate Variability (HRV)

The procedure for recording HRV was thoroughly explained to all the subjects. A minimum of two hours gap was kept between the last meal and the procedure of recording HRV. The subject was told to abstain from any caffeinated drinks and vigorous physical activity prior to recording of HRV. Lead II ECG was recorded for 5 minutes following 15 minutes of relaxation in the supine position for all the subjects. The test was conducted in a lab that was quiet, well ventilated and adequately lit.

Name of the Equipment: AD Instruments Power Lab 8/35 having high performance data acquisition system with Lab Chart 8 software for windows was used for acquisition of HRV recordings.

The following HRV parameters were recorded in each subject:

Time Domain Analysis of HRV

SDNN: Standard deviation of the normal to normal beat R-R interval.

RMSSD: Root mean square of the standard deviations of the normal to normal beat R-R intervals.

pNN50: Proportion of normal to normal beat R-R intervals > 50 milliseconds out of total normal to normal beat R-R intervals.

Frequency Domain Analysis of HRV

LF: Low Frequency power (0.04 – 0.1 Hz)

HF: High Frequency power (0.15-0.4) - Mainly depicts the parasympathetic component

LF / HF Ratio: depicts the sympathy- vagal balance.

Statistical Analysis

The data obtained was statistically analyzed in Microsoft excel sheet using Epi Info version 7. Comparison of data between the two groups was done using the Student 't' and 'chi square test'. Pearson's correlation was used to study the correlation between anthropometric parameters and indices of HRV. Level of significance was set at 5%.

Observations and Results

This study was conducted in the department of Physiology, of a Government Medical College located in Delhi/NCR region. The aim of the study was to study the relationship between obesity the and HRV parameters of young Indian adults. Total 60 subjects were included, divided into two groups of 30 each. The mean age of the subjects was comparable in the two groups (Table 1).

Table 1: Age distribution of the subjects

Groups	Mean age	Std. Deviation	P value
Group I	19.97	0.72	0.27
Group II	20.23	1.10	
Total	20.10	0.93	

The sex distribution of the subjects can be seen in Table 2. The number of males and females were comparable in the two groups.

Table 2: Sex distribution of the subjects

Sex	BMI		Total
	Group I	Group II	
Male	17	16	33
	56.7%	53.3%	55.0%
Female	13	14	27
	43.3%	46.7%	45.0%
Total	30	30	60

The anthropometric profile of the subjects can be seen in Table 3. All the anthropometric parameters except height

Table 3: Anthropometric profile of the study population

Groups		Weight (Kg)	Ht. (cms)	NC (cms)	WC (cms)	HC (cms)	BMI (Kg/m ²)	W/H ratio	Visceral Body Fat
I	Mean	61.13	168.17	35.33	77.80	92.93	21.48	0.84	497.32
	SD	9.85	9.45	3.65	6.57	5.65	1.88	0.06	674.92
II	Mean	81.24	168.80	36.95	97.48	108.26	28.40	0.89	2797.38
	SD	12.68	7.98	3.30	10.30	6.64	2.63	0.05	997.708
P value		<0.001* *	0.78 (NS)	0.08 (NS)	<0.001**	<0.001**	<0.001**	<0.001**	<0.001**

** - 'P' value < 0.001 – highly significant

NS – 'P' value > 0.05 – not significant

Table 4: Time domain analysis of HRV of the subjects in the two groups

Groups		SDNN (ms)	RMSSD (ms)	pNN50 (%)
Group I	Mean	59.873	51.00567	33.44
	Std. Deviation	15.049	22.264	18.53
Group II	Mean	48.2887	38.89477	21.6846
	Std. Deviation	19.31830	18.343926	21.02340
P value		0.001**	0.02*	0.02*

*- 'P' value < 0.05 – significant

** - 'P' value < 0.001 – highly significant

Table 5: Frequency domain analysis of HRV of the subjects in the two groups

Groups		LF (nu)	HF (nu)	LF/ HF ratio
Group I	Mean	43.103	57.369	1.001897
	Std. Deviation	12.112	12.112	1.20899
Group II	Mean	53.3907	45.6833	1.717033
	Std. Deviation	19.34059	18.51227	1.6064175
P value		0.001**	0.02*	0.03*

**-. 'P' value < 0.001 – highly significant

*- 'P' value < 0.05 – significant

Table 6: Correlation of anthropometric parameters with HRV in the two groups

Anthropometry	HRV	Correlation coefficient	'P' value
BMI (Kg/m ²)	SDNN	0.003	0.98 (NS)
	RMSSD	-0.050	0.71 (NS)
	pNN50	-0.561	<0.001**
	LF	0.069	0.60 (NS)
	HF	-0.087	0.51 (NS)
	LF:HF	0.114	0.38 (NS)

and neck circumference (NC) were significantly higher in Group II as compared to Group I.

The time domain parameters of HRV of the two groups can be seen in Table 4. Significant rise in SDNN (ms), RMSSD (ms) and pNN50 in the normal weight subjects was found when compared with obese subjects signifying a higher parasympathetic activity in them.

The Frequency Domain Analysis of HRV of the subjects in the two groups can be seen in Table 5. Significant rise in LF (nu) and LF/ HF ratio was found in group II in comparison to Group I signifying increased sympathetic activity in the obese. HF (nu) was significantly higher and LF (nu) and LF/HF ratio were significantly lower in group I as compared to group II.

NC (cms)	SDNN	0.015	0.91 (NS)
	RMSSD	-0.014	0.92 (NS)
	pNN50	-0.281*	0.03*
	LF	0.266*	0.04*
	HF	-0.285*	0.02*
	LF/HF	0.230	0.07 (NS)
WC (cms)	SDNN	-0.103	0.43 (NS)
	RMSSD	-0.128	0.33 (NS)
	pNN50	-0.502**	< 0.001**
	LF	0.122	0.35 (NS)
	HF	-0.129	0.32 (NS)
	LF/HF	0.141	0.28 (NS)
HC (cms)	SDNN	-0.131	0.31 (NS)
	RMSSD	-0.155	0.24 (NS)
	pNN50	-0.549**	<0.001**
	LF	0.182	0.16 (NS)
	HF	-0.195	0.13 (NS)
	LF/HF	0.208	0.11 (NS)
W/H ratio	SDNN	-0.019	0.88 (NS)
	RMSSD	-0.023	0.86 (NS)
	pNN50	-0.236	0.06 (NS)
	LF	0.002	0.98 (NS)
	HF	-0.001	0.99 (NS)
	LF/HF	0.016	0.90 (NS)
Visceral body fat	SDNN	-0.067	0.61 (NS)
	RMSSD	-0.102	0.44 (NS)
	pNN50	-0.541**	< 0.001**
	LF	0.112	0.39 (NS)
	HF	-0.124	0.34 (NS)
	LF/HF	0.140	0.28 (NS)

** - 'P' value < 0.001 – highly significant

* - 'P' value < 0.05 – significant

NS – 'P' value > 0.05 – not significant

Correlation of the various anthropometric parameters with parameters of HRV in the two groups can be seen in Table 6. pNN50 was seen to be significantly negatively correlated to all anthropometric parameters except W/H ratio. Neck circumference was seen to be significantly negatively correlated with HF (nu) and positively correlated with LF (nu).

Discussion

Our population based cross-sectional study reveals the change of HRV spectrum in accordance with obesity in young healthy Indian adults. A total of 60 subjects were selected on the basis of pre-defined inclusion and exclusion criteria and were divided into two groups of 30 each: Group 1 – Normal weight subjects (BMI – 18 to 22.9) and Group 2 – subjects with BMI \geq 25.

The various anthropometric parameters except height and neck circumference measured in both groups showed significantly higher values in obese subjects as compared to those having normal BMI. The different parameters of HRV were found to be deranged in the obese as compared to the controls.

The efferent vagal activity is a major contributor to the HF component in spectral analysis and SDNN and RMSSD in time domain analysis, as seen in clinical and experimental observations and is an important determinant of cardiovascular health. In our study it is found that there is statistically significant decrease in SDNN, RMSSD, pNN50 and HF (nu) in obese individuals, which is indicative of poor vagal control in the cardiovascular system. A decreased parasympathetic activity is a hall-mark for the obese group of people indicating early cardiovascular vagal tone changes in them. This finding is consistent with a similar study done by Gutin et al. in the year 2014.²²

The obese group were found to have statistically significant increased resting sympathetic activity as evidenced by increase in LF (nu) and LF/HF Ratio in frequency domain analysis of HRV in comparison to the control group.

In our study, LF/HF ratio was also found to be significantly increased in obese subjects compared with normal ones indicating association of obesity with cardiovascular sympathovagal imbalance.

Therefore, the major findings of this study i.e. \downarrow SDNN, \downarrow RMSSD, \downarrow pNN50 and \downarrow HF(nu) components of HRV in cases as compared to controls is effective in demonstrating

impaired parasympathetic activity and significantly \uparrow LF(nu) component and \uparrow LF/HF ratio shows elevated level of sympathetic activity in obese cases compared to controls thus confirming the high prevalence of alterations in HRV in obese healthy, young individuals. Though this study is in contrast to some other studies, which reported that, if the obesity lasts for a longer duration, then it leads to an overall reduction in autonomic activity along with a reduction in sympathetic activity¹¹ also. The high prevalence of alteration in HRV in obese individuals in comparison to normal subjects has been demonstrated by a number of similar studies done earlier.^{12-20,4} Similar findings as ours have also been reported by Thorp and Schlaich,²³ Laederach-Hofmann et al²⁴ and Esler et al.²⁵

The exact mechanism that may cause derangement of HRV in obese has not yet been clearly established. Obesity has been positively established as a state of impaired glucose tolerance leading to 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.²⁶ Thus, hyperinsulinemia may contribute to low cardiac vagal activity.²⁷ Various other studies²⁸⁻³¹ also support similar assumption. The autonomic profiling may thus help us in identifying obese patients at increased risk for cardiovascular disorder.³²

Another hypothesis given for a lower SDNN observed in present study is diminished baroreceptor reflex modulation of RR intervals³³ resulting in low SDNN and low HF power which taken together are indicative of poor vagal control in the cardiovascular system.

The role of the adipokines in HRV is attracting much attention. Low serum concentrations of adiponectin, an adipokine considered as a protective cardiovascular factor, was associated with sympathovagal imbalance favoring relative sympathetic activation in patients with type-2 diabetes.³⁴ Leptin, a strong correlate of the degree of obesity has been found to stimulate the sympathetic nervous system¹⁸⁻¹⁹ and the relation between serum leptin levels and sympathetic activity independently of the amount of observed body fat has also been confirmed.³⁵ Resistin, an adipokine potentially linked to atherogenesis³⁶, has not yet been demonstrated to be linked with the autonomic nervous system.

It is estimated that as much as one-third of the cases of hypertension may be attributable to obesity.³⁷ Higher sympathetic activity has also been related to a higher susceptibility to fatal arrhythmia and development of coronary artery disease (CAD).³⁸ It has also been demonstrated to be the strongest independent predictor of the progression of focal coronary atherosclerosis.³⁹ In a study conducted using a tilt table it was found that HRV values improve after treatment for weight reduction for three months.⁴⁰ Regular assessment of HRV parameters can therefore be used as a biomarker and screening tool for detecting latent cardiac autonomic dysfunction in individuals who are overweight but yet asymptomatic.

Correlation analysis between the anthropometric indices and HRV indices in this study showed that the pNN50 values of time domain indices of HRV was significantly correlated negatively with all anthropometric parameters except W/H ratio. NC was significantly negatively correlated with HF (nu) and positively correlated with LF (nu). Other parameters of obesity however failed to show significant correlation with HRV the reason for which could be the smaller sample size of our study. Various other studies have showed stronger correlation between obesity parameters and HRV than ours,^{4,14,16,41} the reason for which could be larger sample size of their studies. However, Yadav et al. have reported no significant correlation between obesity and HRV parameters despite recording a significant deterioration in HRV in obese subjects compared to normal subjects.¹⁵

Thus it can be said that obese persons are liable to suffer from an increased mortality risk due to cardiovascular disorders due to either continuously lowered parasympathetic or increased sympathetic activity. Thus, early interventional programs and lifestyle approaches for reduced-calorie consumption, high lean body mass, and weight loss approaches like life style changes, diet modification, yoga, stress management and physical exercises, which reduce fat content of the individual, can be advised to obese subjects to reduce the chances of subsequent cardiac morbidity and mortality. Hence, further studies in larger population groups need to be undertaken to validate these inferences. Also, longitudinal studies can help to determine how weight loss affects HRV parameters modifying cardiovascular risks in later life.

Limitation of the Study

The major limitation of our study was the small sample size (30 in each group) preventing broad generalization of the results to the larger population. This was due to restriction of the ICMR project to be conducted over a period of two months only. Metabolic status and insulin resistance of the subjects were not measured which could have given an account of the metabolic activity in obese persons that could be responsible for the HRV changes in them. Influence of ethnicity was not considered in the present study which affects both the anthropometric parameters as well as the indices of heart rate variability in an individual.

Conclusion

Our study detected higher sympathetic and lower parasympathetic activity in obese group compared to normal weight subjects as seen in the Heart Rate Variability analysis. This study helps us to improve our understanding of how obesity affects cardiac autonomic activity, which is an important indicator of cardiovascular morbidity and mortality so that weight reduction, diet modification and appropriate lifestyle interventions like physical exercise and Yoga can be introduced in obese subjects at an early stage in life to prevent cardiovascular mortality from setting in.

Conflict of Interest: Nil.

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. Chhatwal J, Verma M, Riar SK. Obesity among preadolescent and adolescents of a developing country. *Asia Pac J Clin Nutr* 2004;13(3):231-5.
3. Mohan B, Kumar N, Aslam N, Rangbulla A, Kumbkarni S, Sood NK, et al. Prevalence of Sustained Hypertension and Obesity in Urban and Rural School Going Children in Ludhiana. *Indian Heart J* 2004;56:310-4.
4. Rajalakshmi R, VijayaVageesh Y, Nataraj SM, Murali Dhar, Srinath CG. Heart rate variability in Indian obese young adults. *Pak J Physiol* 2012;8(1):39-44.
5. <http://timesofindia.indiatimes.com/india/Indians-adding-to-world-obesity-problem/Report/articleshow/28395856.cms>
6. Vinik AI, Maser RE, Mitchell BD, Freeman R. Diabetic Autonomic Neuropathy. *Diabetes Care* 2003;26(5):1553-79.
7. Kleiger RE, Miller JP, Bigger JT Jr, Moss AJ. The Multicenter Post-Infarction Research Group. Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *Am J Cardiol* 1987;59:256-62.
8. Bigger JT Jr, Fleiss JL, Steinman RC, Rolnitzky LM, Kleiger RE, Rottman JN. Frequency domain measures of heart period variability and mortality after myocardial infarction. *Circ* 1992;85:164-71.
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. *Eur Heart J* 1996;17(3):354-81.
10. Hayano J, Sakakibara Y, Yamada A, Yamada M, Mukai S, Fujinami T. Accuracy of assessment of cardiac vagal tone by heart rate variability in normal subjects. *Am J Cardiol* 1991;67(2):199-204.
11. Masuo K, Mikami H, Ogihara T, Tuck ML. Weight gain-induced blood pressure elevation. *Hypertens* 2000;35:1135-40.
12. Sarkar S, Borade SG, Mukherjee S, Jadhav AA, Saha S. A study of heart rate variability in underweight and overweight young individuals. *Indian J Physiol Allied Sci* 2015;69(3):76-82.
13. Garg N, Gupta P, Verma P, Jain N, Mittal S. BMI based study of heart rate variability in young adults. *Int J Basic Appl Physiol* 2016;5(1):182-6.
14. Lutfi MF, Sukkar MY. Relationship of height, weight and body mass index to heart rate variability. *Sudan Med J* 2011;47(1):14-19.
15. Yadav RL, Yadav PK, Yadav LK, Agrawal K, Sah SK, Islam Md N. Association between obesity and heart rate variability indices: an intuition towards cardiac autonomic regulation – a risk of CVD. *Diabetes, Metabolic Syndrome and Obesity. Targets Ther* 2017;10:57-64.
16. Bhat R, Ganaraja B, Meenu S, Nayanatara AK, Deviprasad S. A study of correlation of anthropometric parameters and heart rate variability among medical students in South India. *Int J Appl Biol Pharm Technol* 2013;4(2):160-5.
17. Pramodh V, Prashanth Kumar M, Prasad BAK. Heart rate variability in obese individuals. *IOSR J Dent Med Sci* 2014;13(5):41-5.
18. Windham BG, Fumagalli S, Alessandro Ble A, Sollers JJ. The relationship between heart rate variability and adiposity differs for central and overall adiposity. *J Obes* 2012;149516.
19. Indumathy J, Pal GK, Pal P, Ananthanarayanan PH, Parija SC, Balachander J, Dutta TK. Association of sympathovagal imbalance with obesity indices and abnormal metabolic biomarkers and cardiovascular parameters. *Obes Res Clin Pract* 2015;9(1):55-66.
20. Rastović M, Srdić-Galić B, Barak O, Stokić E. Association between anthropometric measures of regional fat mass and heart rate variability in obese women. *Nutr Diet* 2017;74(1):51-60.
21. WHO expert consultation. Appropriate body mass index for Asian population and its implications for policy and intervention strategies. *Lancet* 2004;363:157-63.
22. Gutin B, Barbeau P, Litaker MS, Ferguson M, Owens S. Heart rate variability in obese children: relations to total body and visceral adiposity, and changes with physical training and detraining. *Obes Res* 2000;8:12-19.
23. Thorp AA, Schlaich MP. Relevance of sympathetic nervous system activation in obesity and metabolic syndrome. *J Diabetes Res* 2015;2015:Article ID 341583.
24. Laederach-Hofmann K, Mussgay L, Ruddel H. Autonomic cardiovascular regulation in obesity. *J Endocrinol* 2000;164(1):59-66.
25. Esler M, Straznicki N, Eikelis N, Masuo K, Lambert G, Lambert E. Mechanisms of sympathetic activation in obesity-related hypertension. *Hypertens* 2006;48(5):787-96.
26. Van de Borne P, Hausberg M, Hoffman RP, Mark AL, Anderson EA. Hyperinsulinemia produces cardiac vagal withdrawal and nonuniform sympathetic activation in normal subjects. *Am J Physiol* 1999;276:178-83.
27. Valensi P, Paries J, Lormeau B, Attia S, Attali JR. Influence of nutrients on cardiac autonomic function in nondiabetic overweight subjects. *Metab* 2005;54(10):1290-6.
28. Emdin M, Gastaldelli A, Muscelli E, Macerata A, Natali A, Camastra S, Ferrannini E. Hyperinsulinemia and autonomic nervous system dysfunction in obesity: effects of weight loss. *Circ* 2001;103(4):513-9.
29. Martini G, Riva P, Rabbia F, Molini V, Ferrero GB, Cerutti F, Carra R, Veglio F. Heart rate variability in childhood obesity. *Clin Auton Res* 2001;11(2):87-91.
30. Facchini M, Malfatto G, Sala L, Silvestri G, Fontana P, LaFortuna C. Changes of autonomic cardiac profile after a 3-week integrated body weight reduction program in severely obese patients. *J Endocrinol Invest* 2003;26(2):138-42.
31. 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;11(9):1040-7.
32. Peterson HR, Rothschild M, Weinberg CR, Fell RD, McLeish KR, Pfeifer MA. Bodyfat and the activity of the autonomic nervous system. *N Engl J Med* 1988;318(17):1077-83.
33. Muralikrishnan K, Balasubramanian K, Rao B V. Heart rate variability in normotensive subjects with family history of hypertension. *IJPP* 2011;55(3):253-561.
34. Wakabayashi S, Aso Y. Adiponectin concentrations in sera from patients with type-2 diabetes are negatively associated with sympathovagal balance as evaluated by power spectral analysis of heart rate variation. *Diabetes Care* 2004;27:2392-7.
35. Paolisso G, Manzella D, Montano N, Gambardella A, Varricchio M. Plasma leptin concentrations and cardiac autonomic nervous system in healthy subjects with different body weights. *J Clin Endocrinol Metab* 2000;85:1810-4.
36. Burnett MS, Lee CW, Kinnaird TD et al. The potential role of resistin in atherogenesis. *Atheroscler* 2005;182(2):241-8.
37. Huxley R, Mendis S, Zheleznyakov E, Reddy S, Chan J. Body mass index, waist circumference and waist: hip ratio as predictors of cardiovascular risk – a review of the literature. *Eur J Clin Nutr* 2010;64(1):16-22.
38. Schwartz PJ, La Rovere MT, Vanoli E. Autonomic nervous system and sudden cardiac death. Experimental basis and clinical observations for post-myocardial infarction risk stratification. *Circ* 1992;85(1):177-91.

39. Hikuri HV, Jokinen V, Syväne M, Nieminen MS, Alraksinen KEJ, Ikaheimo MJ, et al. Heart rate variability and progression of coronary atherosclerosis. *Atheroscler Thromb Vasc Biol* 1999;19(8):1979–85.
40. Barbara Zahorska-Markiewicz, Katarzyna Mizia-Stec. Tilt table testing in obesity. *Int J Cardiol* 2003;88(1):43–8.
41. Meier U, Gressner AM. Endocrine regulation of energy metabolism: review of pathobiochemical and clinical chemical aspects of leptin, ghrelin, adiponectin and resistin. *Clin Chem* 2004;50:1511–25.

How to cite this article: Khullar S, Sinha P, Singh M, Diksha. Relationship between heart rate variability and anthropometric parameters in young Indian adults. *Indian J Clin Anat Physiol* 2019;6(2):209-15.