ORIGINAL ARTICLE

HEART RATE VARIABILITY IN INDIAN OBESE YOUNG ADULTS

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Background: Obesity which is characterised by haemodynamic and metabolic alterations has become an epidemic problem. In obese children and adults it is shown that there is autonomic dysfunction. The objective of this study was to assess Heart Rate Variability (HRV) in obese Indian young adults and to establish association between HRV indices and obesity indices. **Methods:** This study included 60 young adult subjects in the age group 18–20 years. According to BMI subjects were classified into obese group (BMI ≥25 Kg/m^2) and normal weight group (BMI 18–22.9 Kg/m^2). Obesity indices like Waist Circumference, Hip Circumference, Waist Hip Ratio, Waist Stature Ratio and Body Fat percentage were measured and calculated. ECG was recorded in each subject in lead II for 5minutes using PowerLab® system and analysis was done using HRV module LabChart® 7.1 software. **Results:** There was significant increase in the mean heart rate, LF (nu) and LF/HF ratio whereas SDNN, SDANN, rMSSD, pNN50, Total power, HF(AV) and HF (nu)were significantly lower (p<0.05) in obese group. Pearson's correlation analysis showed significant correlation between HRV and obesity indices. On regression analysis BMI was the major predictor for HRV variations. **Conclusions:** The study demonstrates decrease in overall HRV. The HRV indices indicating sympathetic activity were increased whereas those indicating parasympathetic activity were decreased in obese young adults showing autonomic imbalance in them. BMI was the major predictor for HRV variations.

Keywords: Obesity, Obesity indices, Heart rate variability, Autonomic functions

INTRODUCTION

Obesity is a nutritional health problem which is gradually increasing and affecting all sections of population *viz* children, adolescents and adults**.** According to WHO documents one billion people are overweight and 300 million are obese worldwide. Epidemiological data shows that prevalence rates are increasing not only in industrialised countries but also in developing countries especially in the adolescent population. Obesity is on rise in our society due to socioeconomic developments leading to change in lifestyle particularly dietary pattern.[1,2] Obesity is characterised by haemodynamic and metabolic alterations. Obese individuals have higher prevalence of many diseases like coronary heart disease, hypertension, hyperlipidemia and diabetes mellitus. All these diseases are preventable by changes in lifestyle of a person.

The autonomic nervous system plays an important role, not only in physiological situations, but also in various pathological settings such as diabetic neuropathy, myocardial infarction and congestive heart failure. Autonomic imbalance with increased sympathetic activity and reduced vagal tone has been strongly implicated in the pathophysiology of development of arrhythmias and sudden cardiac deaths.[3–5] 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 sinoatriallevel.[6]

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. 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[7]and sudden cardiac death.[8] 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. High frequency power is mediated by vagal activity,[6,9] 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 modulations.[6,10,11]

It is shown that in obese people, there is reduction in the HRV reflecting a predominant sympathetic influence on control of cardiac functions. HRV studies among adults and children with obesity have revealed inconsistent results including high[12,13] and low[14] sympathetic tones coupled with a reduction in vagal tone.[12,14] It has also been shown in western studies on obese adults that weight loss reverses back to parasympathetic control of cardiac functions.[15] While there are 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 under taken with the aim of evaluating the resting cardiac autonomic nerve activity given by the changes in HRV analysis in healthy obese young adults.

Specific objectives of the study were: a) To know the heart rate and HRV changes in healthy obese young adults; b) To compare the same with that in normal weight subjects; and c) To assess the role of different obesity indices in predicting the HRV changes.

MATERIAL AND METHODS

This cross-sectional study includes participants from first year medical students (n=150) of JSS Medical College, Mysore. They were requested to complete a questionnaire that included specific information on age, history of hypertension, cardiac or pulmonary diseases, tobacco and alcohol intake and physical activity. Those in the age group of <18 year and >22 year (n=2), doing regular exercise, with the history of hypertension (n=2) and pulmonary disease (n=1) were excluded from the study. Clinical examination was conducted on all subjects to rule out any systemic disorders. Thus 145 students were available for the study satisfying the inclusion and exclusion criteria. Weight to the nearest 0.5 Kg, Height, Waist circumference (WC) and Hip Circumference (HC) to the nearest 0.1 Cm were measured for each student according to standard procedures. Body Mass Index (BMI), Waist Hip Ratio (WHR) and Waist Stature Ratio (WSR) were then calculated. BMI was calculated by dividing weight in Kg by the square of the height in meters and the two WHR and WSR were calculated by performing appropriate divisions. Body Fat Percentage (BF%) was calculated using equation to predict percentage body fat from BMI.[16]

BF%=63.7-864×(1/BMI)-12.1×Sex+0.12×Age+129×Asian ×(1/BMI)-0.091×Asian×Age-0.030×African American×Age

Where, Sex=1 for male and 0 for female; Asian=1 and 0 for other races; African American=1 and 0 for other races; Age in yrs; BMI in Kg/m^2

Subjects were divided into two groups depending on their BMI, Obese group with BMI ≥25 Kg/m^2 (n=30) (male=17 and female=13) was considered to form the study group. Out of 115 Normal weight students 30 (male=17 and female=13) were selected randomly using random number table. Thus the study consisted of two groups namely Obese and Normal weight with 30 subjects in each group. This sample size was estimated to be enough to detect a clinically relevant difference of 10% in the HRV parameters at 5% level of significance with 80% power.

The study was carried out in the Human Physiology Laboratory of Department of Physiology JSS Medical College, Mysore between 3 to 5 PM. After reporting, the subjects were asked to relax for 15 minutes in supine posture. Then ECG was recorded for 5 minutes in lead II in supine position and breathing normally. ECG was recorded using Powerlab® 8/30, ML870 high performance data acquisition system with LabChart® 7.1 software for windows. Signal acquisition, storage and processing were performed on computer. The digitised ECG signals were analyzed on-line and simultaneously stored on removable hard disks for off-line verification. HRV analysis of ECG recordings was carried out using HRV module software for LabChart®. HRV module analyses beat to beat interval variation in ECG recordings by detecting the R waves from each ECG waveform and generating an R-R interval data for analysis.

HRV analysis included time and frequency domain indices. Time domain indices in milliseconds includes SDNN– Standard Deviation of the all NN interval, SDANN– Standard Deviation of the average NN intervals calculated over short periods, usually 5 minutes, rMSSD– square root of the mean of the sum of the squares of differences between adjacent NN interval, NN50– adjacent NN intervals that are greater than 50 ms, pNN50(%)– percentage of difference between adjacent NN intervals that are greater than 50 ms. Frequency domain indices includes TP ($msec^2$)– Total power, variance of all NN intervals in $msec^2$, VLF ($msec^2$)– very low frequency band variation, LF (AV) absolute values in $msec^2$ and in normal units (nu)– low frequency band variation, HF-AV in $msec^2$ and in nu– High frequency band variation, LF/HF ratio.

The study was approved by the Institutional Ethical committee of JSS Medical College, Mysore. Subjects were explained about the purpose of the study, the study protocol and the informed consent was obtained.

Statistical Analysis:

Statistical analysis were performed using Microsoft excel and SPSS 17 software. In order to compare physical characteristics, time domain and frequency domain of HRV indices between the groups mean and standard deviation of the same were estimated and the significance of the difference between the two groups was tested by applying Student's *t*-test for independent samples. Pearson's correlation coefficients were estimated to quantify the linear relationship between the indices of obesity and HRV

indices. Subsequently the significant indices of obesity were selected to fit the linear regression on HRV indices. Simple linear regressions were fit to know the role of individual parameters followed by multiple regressions to find the role of significant parameters collectively in prediction of HRV indices.

RESULTS

The present study included 60 subjects (30 in obese group and 30 in Normal Weight groups) in the age group of 18–22 years. The Physical characteristics of the two groups are shown in Table-1. There was no significant difference in the mean of age and height between the groups. There was significant difference in the mean of weight, WC, HC, WHR, WSR, BMI and percentage of body fat ($p<0.05$). WC in obese males and females was 94.27±9.04 and 87.04±7.17 respectively, these values were more than the cut-off points for Indian population (cut-off points for male: >90 Cm and females: >80 Cm). The heart rate was significantly higher in obese group in comparison with normal weight group as shown in Table-2a.

Time domain indices of HRV (Table-2a):

SDNN (ms), SDANN (ms), rMSSD (ms) and pNN50 (%) in obese group were significantly decreased when compared to normal weight group ($p<0.05$). NN50 (ms) was also less in obese group when compared to normal weight group however the difference was not statistically significant ($p>0.05$).

Pearson's correlation coefficient showed that SDNN, SDANN and rMSSD were negatively correlated with BMI and WSR which was statistically significant. They also showed negative correlation with WC, HC, WHR and BFP which was however, not statistically significant. pNN50 showed negative correlation with BMI, WC, WHR, WSR and BFP, however, it was not statistically significant. On simple linear regression analysis for those time domain indices which were significantly correlated with obesity indices, BMI explained 9.7% of variation in SDNN, 9.4% variation in SDANN and rMSSD, whereas WSR explained 6.5% of variation in SDNN and 11.4% variation in SDANN and rMSSD. On multiple regressions, it was found that, the predictive value of BMI and WSR taken together was 10–11% in prediction of any of the HRV indices.

Frequency domain indices of HRV (Table-2b)**:**

TP, HF (AV) and HF (nu) were significantly lower in obese group when compared to normal weight group. VLF was also lower in obese group which was not statistically significant. LF (nu)– low frequency power in normalised units and LF/HF ratio were significantly higher in obese group. LF (AV) was also higher in obese group but was not statistically significant.

Pearson's correlation coefficient showed that all the indices of obesity except BFP were significantly correlated with Frequency indices of HRV. HF (AV) and HF (nu) were negatively correlated where as LF (nu) and LF/HF ratio were positively correlated. TP also showed negative correlation however it was not statistically significant. On simple linear regression analysis for these frequency domain indices which were significantly correlated with obesity indices, the percentage variation explained by different obesity indices individually varied from 8 to 12 in case of LF (nu), 9 to 17 for HF (AV), 8 to 11 for HF (nu) and 10 to 15 for LF/HF ratio. By and large, WSR was most important individual parameter in prediction of the HRV indices followed BMI. In multiple regression analysis, it was found that the significant obesity indices taken together were responsible for 24 percent of variation in case of HF (AV) followed by 17% in case of LF (nu) and LF/HF ratio and 12% in case of HF (nu).

Table-1: Physical Characteristics of the two groups

Parameters	Normal weight (Mean±SD)	Obese (Mean±SD)	*p*-value
Age (Year)	18.4±0.61	18.1±0.80	0.155
Weight (Kg)	57.8±8.04	80.4±12.35	<0.001
Height (m)	1.68±0.11	1.67±0.07	0.775
BMI (Kg/m^2)	20.4±1.37	28.6±3.06	<0.001
BFP (%)	20.7±5.93	31.0±6.20	<0.001
Waist C (Cm)	76.0±6.60	92.2±10.14	<0.001
Hip C (Cm)	92.0±4.20	105.7±8.61	<0.001
WHR	0.83±0.05	0.87±0.05	<0.001
WSR	0.45±0.04	0.55±0.05	<0.001

Table-2a: Heart rate and Time domain indices of HRV for the two groups

Parameters	Normal weight (Mean±SD)	Obese (Mean±SD)	*p*-value
HR (bpm)	79.3±8.36	84.3±9.48	0.033
SDNN (msec^2)	70.1±21.22	54.8±17.16	0.003
SDANN (msec^2)	57.1±23.35	40.4±18.03	0.003
rMSSD (msec^2)	57.0±23.28	40.3±17.99	0.003
NN50 (msec^2)	76.0±41.53	60.5±49.29	0.193
PNN50 (%)	28.1±15.88	18.9±15.10	0.025

Table-2b: Frequency domain indices of HRV between the two groups with p-value for significance of difference.

Parameters	Normal weight (Mean±SD)	Obese (Mean±SD)	*p*-value
TP (msec^2)	4824.6±1864.58	3847.2±1555.40	0.032
VLF (msec^2)	1333.8±806.80	979.2±660.49	0.068
LF(AV) (msec^2)	1862.8±919.75	1975.7±1118.75	0.671
LF (nu)	48.9±17.85	59.5±15.58	0.018
HF(AV) (msec^2)	1535.7±916.62	841.2±576.14	0.0009
HF (nu)	41.4±15.19	32.2±12.15	0.013
LF/HF	1.2±0.64	2.4±1.69	0.001

Table-3: Pearson's correlation coefficient between Obesity indices and Time and Frequency domain indices of HRV

Parameters	BMI	WC	HC	WHR	WSR	BFP
HR	0.08	0.05	0.04	0.05	0.14	0.12
SDNN	-0.31*	-0.16	-0.14	-0.15	-0.25*	-0.24
SDANN	-0.34**	-0.22	-0.20	-0.20	-0.31*	-0.21
rMSSD	-0.34**	-0.22	-0.20	-0.20	-0.31*	-0.21
NN50	-0.04	0.02	0.09	-0.13	-0.07	-0.11
pNN50	-0.19	-0.10	-0.08	-0.13	-0.20	-0.18
TP	-0.22	-0.16	-0.10	-0.21	-0.18	-0.13
VLF	-0.09	0.02	-0.03	-0.04	-0.04	-0.19
LF(AV)	0.03	0.03	0.08	0.03	0.04	0.17
LF (nu)	0.32*	0.35**	0.28*	0.36**	0.33**	0.01
HF(AV)	-0.40**	-0.37**	-0.30*	-0.37**	-0.41**	-0.21
HF (nu)	-0.32*	-0.28*	-0.24	-0.24	-0.33**	-0.15
LF/HF	0.39**	0.34**	0.31*	0.28*	0.39**	0.19

*p<0.05, **p<0.01

Table-4a: Results of Simple linear regression analysis: Regression coefficient (β), Variation explained (R^2) and p-value for significance of β

Dependent variable	Predictor	β	R^2(%)	p-value
SDNN	BMI	- 1.35	9.70	0.02
	WSR	- 81.57	6.50	0.049
SDANN	BMI	-1.58	11.40	0.008
	WSR	-106.27	9.40	0.02
rMSSD	BMI	-1.57	11.40	0.008
	WSR	-106.01	9.40	0.02
LF(nu)	BMI	1.16	10.00	0.014
	WC	0.51	12.00	0.007
	HC	0.50	7.60	0.033
	WHR	117.50	7.50	0.033
	WSR	90.36	11.00	0.009
HF(AV)	BMI	-70.78	16.20	0.001
	WC	-26.43	13.80	0.003
	HC	-26.39	9.30	0.018
	WHR	-5847.40	13.80	0.003
	WSR	-5315.09	16.70	0.001
HF(nu)	BMI	-0.95	9.90	0.014
	WC	-0.34	7.60	0.033
	WSR	-73.97	10.90	0.009
LF/HF ratio	BMI	0.11	15.20	0.002
	WC	0.04	11.60	0.008
	HC	0.04	9.60	0.016
	WHR	7.18	12.80	0.005
	WSR	8.44	15.20	0.002

Table-4b: Results of Multiple regression analysis: percent variation explained (R^2%) and p-value for significance

Dependent Variables	Predictors	R^2(%)	p-value
SDNN	BMI, WSR	10	0.044
SDANN	BMI, WSR	11	0.032
rMSSD	BMI, WSR	11	0.032
LF(nu)	BMI, WSR, WC, HC, WHR	17	0.067
HF(AV)	BMI, WSR, WC, HC, WHR	24	0.010
HF(nu)	BMI, WSR, WC	12	0.073
LF/HF	BMI, WSR, WC, HC, WHR	17	0.065

DISCUSSION

During normal sinus rhythm, the heart rate varies from beat to beat. Heart rate variability (HRV) results from the interplay between the various physiological mechanisms that regulate the heart rate. Since short-term heart rate regulation is predominantly governed by sympathetic and parasympathetic neural activity, by examination of heart rate fluctuations normal functioning of the autonomic nervous system can be tested. There are different measures of HRV– Time domain indices, Frequency domain indices and also non-linear techniques. Time domain analysis of heart rate variability uses statistical methods to quantify the variation of the standard deviation or the differences between successive RR intervals. Frequency domain analysis of heart rate variability enables us to calculate the respiratory dependent high frequency variations.[6,17]

The present study shows that the heart rate was significantly higher in obese young adults. Higher heart rate is a marker of relative sympathetic dominance and is an independent marker of mortality in a wide spectrum of conditions.[18] The study also shows that in obese young adults SDNN, SDANN and TP were significantly lower indicating decrease in overall HRV in young adults. Thus the study confirms the high prevalence of alterations in HRV in obese individuals as shown in earlier studies on obese children,[19] adolescents[20] and adults.[21,22] Reduced heart-rate variability is a marker of sympatho-vagal imbalance.[6] Time-domain measures of heart-rate variability, SDNN and SDANN reflect both sympathetic and parasympathetic modulation of heart rate and reduced SDANN and SDNN values usually indicate relative sympathetic dominance.[23] Furthermore, in prospective studies reduced heart-rate variability has been shown to be the strongest independent predictor of the progression of focal coronary atherosclerosis.[24] Decreased HRV could partly account for the higher cardiovascular risk and incidence of sudden death[8] in obese persons.

Our results suggests that in obese young adults there was decrease in rMSSD, NN50, pNN50 of time domain indices and HF component of frequency domain indices which mainly assess the parasympathetic activity to heart indicating lower parasympathetic activity in obese young adults. These findings are in similar to other studies which demonstrated that obese persons have decreased parasympathetic nerve activity.[12,14,25] 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.[26,27] Thus, hyperinsulinemia may contribute to low cardiac vagal activity.[28]

The present study also shows that obese young adults have higher LF and LF/HF ratio components of frequency domain indices which mainly measure the sympathovagal balance to heart reflecting an increase in sympathetic nerve activity in these persons. The LF/HF ratio has been proposed to be an accurate measure of the overall sympathovagal balance of the autonomic nervous system in which higher values indicate a more sympathetically driven cardiovascular system.[19,29] Earlier studies on sympathetic nerve activity in obese persons have produced conflicting results. Some studies have shown decrease[14,30,31] and some increase in sympathetic activity in obesity.[12,13,32] Measurements of plasma and urinary catecholamine concentrations as indices of sympathetic nervous system activity have ranged from low through normal to high.[14,33,34] Sympathetic nerve activity in skeletal muscle was increased in obese subjects but skin sympathetic nerve activity was not significantly different.[35] Insulin and leptin levels are elevated in obesity. Thus increased insulin and leptin levels are thought to increase sympathetic nervous system activity.[20,36,37]

Correlation analysis between the obesity indices and HRV indices in this study showed that the time domain indices of HRV was significantly correlated negatively with overall obesity measured by BMI and WSR than the central obesity measured by WC, HC and WHR. In frequency domain indices LF component and LF/HF ratio were positively correlated whereas HF component was negatively correlated with both overall as well as central obesity indices. BFP was also negatively correlated with overall HRV and HF component which was not significant. There was also weak positive correlation between BFP and LF component, LF/HF ratio. Age and sex matched regression analysis showed that BMI was the major determinant for the changes in both time domain and frequency domain indices. WSR was the next important predictor for the time domain indices whereas for the frequency domain indices it was WC and WHR.

Obese persons may suffer from an increased mortality risk due to cardiovascular disorders related to either continuously lowered parasympathetic or altered sympathetic activation. Early detection and management by weight reduction and regular exercise can reduced the risk as these are shown to increase HRV.[15,38,39] HRV analysis can detect changes even before clinical signs appear.[6] Thus regular assessment of HRV measures can be used as a biomarker for early detection and subsequent management of cardiovascular diseases in obese individuals.

We acknowledge some of the limitations in our study. The primary limitation of the work presented is of small sample size. Thus broad generalisations of the results to larger populations cannot be done. Secondly the metabolic status and insulin resistance were not measured which could have given an account of metabolic activity in obese persons which could be responsible for HRV changes in them.

CONCLUSION

The present findings demonstrate decreased HRV, higher sympathetic and lower parasympathetic nerve activity in obese subjects. HRV indices were significantly associated with obesity indices. BMI was the major determinant for the changes in both time and frequency domain indices. Thus time and frequency domain analysis of the HRV in obese young adults shows imbalance in the autonomic neural activities to the heart.

REFERENCES

1. Chhatwal J, Verma M, Riar SK. Obesity among pre-adolescent and adolescents of a developing country (India). Asia Pac J Clin Nutr 2004;13(3):231–5.
2. 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.
3. Vinik AI, Maser RE, Mitchell BD, Freeman R. Diabetic Autonomic Neuropathy. Diabetes Care 2003;26(5):1553–79.
4. 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.
5. 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. Circulation 1992;85:164–71.
6. 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.
7. 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.
8. Moolgard H, Sorensen K, Bjerregaard P. Attenuated 24-h heart rate variability in apparently healthy subjects, subsequently suffering sudden cardiac death. Clin Autonom Res 1991;1:233–7.
9. 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.
10. 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.
11. Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. Circulation 1991;84:1482–92.

12. 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.
13. 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.
14. 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.
15. 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.
16. McArdle WD, Katich FI, Katch VL. Body composition, Obesity and Weight Control. In: Essentials of Exercise Physiology, 3rd ed. USA. Lippincott Williams and Wilkins Publications. 2005: 528.
17. Bilchick KC, Berger RD. Heart Rate Variability. J Cardiovasc Electrophysiol 2006;17(6):691–4.
18. Palatini P. Elevated heart rate as a predictor of increased cardiovascular morbidity. J Hypertens 1999;17:S3–S10.
19. Kaufman CL, Kaiser DR, Steinberger J, Kelly AS, Dengel DR. Relationships of Cardiac Autonomic Function With Metabolic Abnormalities in Childhood Obesity. Obesity 2007;15:1164–71.
20. Guízar JM, Ahuatzin R, Amador N, Sánchez G, Romer G. Heart Autonomic Function in Overweight Adolescents. Indian Pediatrics 2005;42:464–9.
21. Zahorska-Markiewicz B, Kuagowska E, Kucio C, Klin M. Heart rate variability in obesity. Int J Obes 1993;17:21–3.
22. Quilliot D, Fluckiger L, Zannad F, Drouin P, Ziegler O. Impaired autonomic control of heart rate and blood pressure in obesity: role of age and of insulin-resistance. Clin Auton Res 2001;11(2):79–86.
23. Lombardi F. Clinical implications of present physiological understanding of HRV components. Cardiac Electrophysiology Rev 2002; 6:245–9.
24. Hikuri HV, Jokinen V, Syvänne 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.
25. Sekine M, Izumi I, Yamagami T, Kagamimori S. Obesity and Cardiac Autonomic Nerve Activity in Healthy Children: Results of the Toyama Birth Cohort Study. Environ Health Prev Med 2001;6(3):149–53.
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:R178–R183.
27. Muscelli E, Emdin M, Natali A, Pratali L, Camastra S, Gastaldelli A, *et al*. Autonomic and hemodynamic responses to insulin in lean and obese humans. J Clin Endocrinol Metab 1998;83:2084–90.
28. Valensi P, Paries J, Lormeau B, Attia S, Attali JR. Influence of nutrients on cardiac autonomic Function in nondiabetic overweight subjects. Metabolism 2005;54:1290–6.
29. Malliani A, Pagani M, Furlan R, Guzzetti S, Lucini D, Montano N, et al. Individual recognition by heart rate variability of two different autonomic profiles related to posture. Circulation 1997;96:4143–5.
30. Akhter S, Begum N, Ferdousi S, Begum S, Ali T. Sympathetic Nerve Function Status in Obesity. J Bangladesh Soc Physiol 2010;5(1):34–9.
31. Colak R, Donder E, Karaoglu A, Ayhan O, Yalniz M. Obesity and the activity of the Autonomic nervous system. Turk J Med Sci 2000;30:173–6.
32. Chen-Chung Fu, Yin-Ming Li, Dee Pei, Chien-Lin Chen, Huey-Ming Lo, Du-An Wu, *et al*. Heart Rate Variability in Taiwanese Obese Children. Tzu Chi Med J 2006;18:199–204.
33. Young JB, Macdonald IA. Sympathoadrenal activity in human obesity: heterogeneity of findings since 1980. Int J Obes 1992;16:959–67.
34. Grassi G, Colombo M, Seravalle G, Spaziani D, Mancia G. Dissociation between muscle and skin sympathetic nerve activity in essential hypertension, obesity, and congestive heart failure. Hypertension 1998;31:64–7.
35. Anderson EA, Balou TW, Hoffman RP, Sinkey CA, Mark AL. Insulin increases sympathetic activity but not blood pressure in borderline hypertensive humans. Hypertension 1992;19:621–7.
36. Gudbjörnsdottir S, Elam M, Sellgren J, Anderson EA. Insulin increases forearm vascular resistance in obese, insulin-resistant hypertensives. J Hypertens 1996;14:91–7.
37. Sjoberg N, Brinkworth GD, Wycherley TP, Noakes M, Saint DA. Moderate weight loss improves heart rate variability in overweight and obese adults with type 2 diabetes. J Appl Physiol 2011;110(4):1060–4.
38. Sandercock GR, Bromley PD, Brodie DA. Effects of Exercise on Heart Rate Variability: Inferences from Meta-Analysis. Med Sci Sports Exerc 2005;37(3):433–9.

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