ORIGINAL ARTICLE

Brief Assessment of Supine Heart Rate Variability in Normal Weight, Overweight, and Obese Females

[Correction added on 2 December 2013, after first online publication: The placements of figures are changed.]

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Background: Little research has been conducted on the heart rate variability (HRV) parameters in late adolescent females. The present study aimed to assess HRV time and frequency domain parameters in overweight and obese late adolescent females. Also to assess any possible correlation between HRV parameters and obesity indices in that particular age group.

Subjects and Methods: Fifteen-minute period of standardized ECG recording was implemented to record HRV time and frequency parameters in 42 normotensive euglycemic female medical students aged (18–21 years); lean ($n = 13$), overweight ($n = 13$), and obese ($n = 16$). For the analysis of results, 2.5-minute data were used.

Results: Root mean squares of successive differences between adjacent RR intervals (rMSSD) and high-frequency (HF) power were significantly decreased in overweight and obese late adolescent females. Parameters reflecting sympathetic activity which include low-frequency (LF) power and LF/HF ratio showed significant increase in overweight group. Interestingly, LF power was significantly reduced in obese group while the LF/HF ratio was insignificantly different. No significant correlations were observed between HRV indices and parameters of total or visceral obesity in the study groups.

Conclusion: HRV indices showed sympathetic hyperactivity in overweight late adolescent females and diminished sympathetic response in matching obese group. Both overweight and obese females showed decreased protective vagal influence on the heart.

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females; heart rate variability; obesity; overweight

INTRODUCTION

Obesity is a serious health problem in both developed and developing countries and it has reached an epidemic proportion in many countries.¹

Obesity is associated with many cardiac complications with increased mortality risks such as coronary heart disease, heart failure, and arrhythmias. It may affect the heart through its influence on known risk factors such as dyslipidemia and hypertension.²

It is believed that female obesity has greatly increased and most of the time it is also followed by related diseases like gout, hypertension, diabetes, cardiac problems, sleep apnea, cancer, osteoarthritis, and many other disorders. It was demonstrated that the prevalence of overweight and obesity

continuously increases in both men and women with higher incidence in women.³

Heart rate variability (HRV), defined as degree of fluctuation of beat-to-beat difference in cardiac rhythm, has been widely used as a functional indicator of autonomic nervous activity to the heart.⁴ HRV has been investigated in girls, adult females, and postmenopausal females,^{5–7} while late adolescent females have not been investigated for the effects of overweight and obesity on HRV. In addition, results of previous investigations regarding the association between HRV and obesity are still conflicting.⁸

AIM OF WORK

This study aimed to assess the HRV time and frequency parameters in overweight, obese, and

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lean late adolescent females and to assess possible correlations between HRV parameters and obesity indices in that particular age group.

SUBJECTS AND METHODS

Subjects

Female volunteer students were recruited from Faculty of Medicine, King Abdulaziz University. Detailed history was obtained to detect exclusion criteria which involved; less than 1-year duration of overweight or obesity, history of metabolic, cardiovascular disease or any acute or chronic illness, participation in any regular physical activity or change in dietary habits during the 6 months preceding the study.

Blood pressure measurements were taken using (Spot Vital Sign, Welch Allyn Inc., Skaneateles Falls, NY, USA). The average of two readings was used to determine the systolic and diastolic values. Forty-two (18–21 years old) normotensive euglycemic participants who fulfilled the criteria were enrolled in the study. The study protocol was approved by the faculty research ethics committee and written informed consents were obtained from all participants. Weight and height measurements were obtained with the participants wearing light clothes and no shoes. Body mass index (BMI) was calculated by dividing weight (kg) by the square of height in (m). Waist hip ratio (WHR) was obtained by dividing the circumference of the abdomen at the level of the umbilicus by that of the hip at the largest perimeter in (cm). Based on the international classification of adult weight according to BMI,⁹ the participants were included into one of the following groups:

Group 1: control lean group; BMI ranged from 18.5 to 24.99 kg/m² (n = 13).

Group 2: overweight group; BMI ranged from 25.00 to 29.99 kg/m² (n = 13).

Group 3: obese group; BMI exceeded 30 kg/m² (n = 16)

Experimental Design

Participants were instructed to visit our lab at day 7 of menstrual cycle to standardize the status of ovarian hormones and avoid introducing a confounding factor.¹⁰

At the day of the experiment, HRV indices were obtained from short-term (15 minutes) electrocardiographic recording while participants were in the supine position.

Electrocardiographic Data Analysis

The ECG recordings were obtained from each participant for 15 minutes. It was sampled at 1000 Hz with the PowerLab acquisition system (AD Instruments Pty Ltd, Castle Hill, Australia) installed on IBM computer. The first 5 minutes of each ECG recording were disregarded to allow for stabilization of the data prior to analysis. The time domain HRV parameters implemented in this study were; the standard deviation of all RR intervals (SDRR), which reflects all the cyclic components responsible for variability in the period of recording, and the root mean squares of successive differences between adjacent RR intervals (rMSSD), which is considered as an index of parasympathetic modulation of HRV.¹¹ Using a sliding window of 64-s duration, time-varying autoregressive modeling of the interpolated RR sequence was performed to estimate its power spectrum (ms²). The order of the time-varying autoregressive model was set to 12. The power spectral analysis included; the very low-frequency power (VLF; 0–0.05 Hz), the low-frequency power (LF; 0.05–0.15 Hz) which is dually regulated by vagal and sympathetic systems,¹² the high-frequency power (HF; 0.15–0.5 Hz) which reflects cardiac vagal activity,¹³ and the total power (0–0.5 Hz). The ratio of LF/HF was calculated to assess the cardiac autonomic balance.¹⁴

Statistical Analysis

Statistical analysis of data was done using SPSS for windows package version 20 (SPSS Inc. Chicago, IL, USA). K-S test was applied to test the normal distribution of various data. One-way analysis of variance ANOVA test was used to compare between means of the variables in the study groups and association between variables was carried out using Pearson's correlation coefficient. A P value ≤0.05 was considered significant.

RESULTS

The physical characteristics of the participants in this study in addition to the mean values of their

Table 1. Physical Parameters of All Groups (Mean $\pm$ SD)

Parameter	Control N = 13	Overweight N = 13	Obese N = 16
Age	19.9 $\pm$ 1.3	19.1 $\pm$ 1.8	19 $\pm$ 1.1
BMI	21.92 $\pm$ 2.36	27.19 $\pm$ 1.71 ^a	36.72 $\pm$ 4.73 ^{ab}
WHR	0.68 $\pm$ 0.04	0.76 $\pm$ 0.07 ^a	0.78 $\pm$ 0.05 ^a
Pulse	69.62 $\pm$ 5.94	70.15 $\pm$ 7.01	69.00 $\pm$ 10.65
SBP	102.31 $\pm$ 10.13	102.31 $\pm$ 9.27	107.05 $\pm$ 10.65
DBP	59.23 $\pm$ 4.94	63.85 $\pm$ 9.61	67.5 $\pm$ 12.38 ^a

^aSignificant when compared to control group.^bSignificant when compared with overweight group.

BMI = body mass index; WHR = waist hip ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure.

Table 2. HRV Parameters of All Groups (Mean $\pm$ SD)

Parameter	Control N = 13	Overweight N = 13	Obese N = 16
SDRR	105.75 $\pm$ 22.82	99.33 $\pm$ 12.63	77.62 $\pm$ 25.14 ^{ab}
rMSSD	131.76 $\pm$ 30.41	96.02 $\pm$ 19.47 ^a	71.24 $\pm$ 21.13 ^{ab}
VLF	1782.85 $\pm$ 495.18	3164.03 $\pm$ 733.32 ^a	770.51 $\pm$ 242.79 ^{ab}
LF	1511.69 $\pm$ 645.95	3669.86 $\pm$ 795.06 ^a	1020.73 $\pm$ 257.76 ^{ab}
HF	2722.71 $\pm$ 649.09	1659.4 $\pm$ 403.39 ^a	1537.47 $\pm$ 497.7 ^a
LF/HF	0.59 $\pm$ 0.3	2.37 $\pm$ 0.82 ^a	0.74 $\pm$ 0.31 ^b

^aSignificant when compared to control group.^bSignificant when compared with overweight group.

SDRR = standard deviation of all RR intervals; rMSSD = root mean squares of successive differences between adjacent RR intervals; VLF = very low-frequency power; HF = high-frequency power; LF = low-frequency power; LF/HF = the ratio of LF/HF.

Figure 1. Scatter plot of heart rate in the study groups. **Figure 2.** Scatter plot of SDRR in the study groups.

pulse rate, systolic and diastolic blood pressures are shown in Table 1 and Fig. 1.

The time and frequency domain parameters of the HRV assessment in the study groups are shown in Table 2.

The rMSSD was significantly decreased in overweight and obese groups when compared to control group ($P < 0.01$, $P < 0.001$), respectively (Figs. 2 and 3).

Figure 3. Scatter plot of rMSSD in the study groups.

Figure 4. Scatter plot of LF waves in the study groups.

The LF were significantly increased in overweight group ($P < 0.001$) and significantly decreased in obese group ($P < 0.05$) when compared to control group (Fig. 4).

Comparing the mean values of HF between the study groups, revealed significant decrease in both overweight and obese groups when compared to control group ($P < 0.001$ for both) (Fig. 5).

The LF/HF ratio showed a significant increase in the overweight group ($P < 0.001$), and was insignificantly different in the obese group when compared to the control group (Fig. 6).

Figure 5. Scatter plot of HF waves in the study groups.

Figure 6. Scatter plot of LF/HF ratio in the study groups.

As regard the correlation studies, the present work found no significant correlations between BMI or WHR and HRV indices in the study groups.

DISCUSSION

This study adds important findings concerning cardiac autonomic regulation in young late adolescent females. The main findings of this study revealed loss of protective vagal influence on heart rate in both overweight and obese late adolescent females. There was also decreased sympathetic influence in obese group; such influence was still

enhanced in overweight group. The present results revealed no correlation between BMI or WHR and HRV parameters. There is consistent evidence that HRV is capable of identifying patient at high risk of all-cause mortality and predicting sudden death.¹⁵

In the current work, the time domain analysis of HRV revealed a significant decrease in rMSSD in overweight and obese late adolescent females compared to their matching lean group. The foregoing data showed evidence of decreased parasympathetic activity in overweight and obese groups. In support of this concept, this study showed a significant decrease in the HF power in overweight and obese females. In accord with our finding, lower parasympathetic modulation of HRV was observed in obese girls with high central fat,⁵ and in obese adults.¹⁶

On the other hand, higher parasympathetic modulation of HRV in obese women was reported specially when there was combination of upper body and visceral obesity.⁶ A favorable vagal HRV profile was also recently observed in obese postmenopausal women.⁷

An explanation for the controversy regarding vagal cardiac influence in overweight and obese females could be attributed to the difference in the age of females in the present and previous researches which ranged between childhood and postmenopause. It seems that cardiac parasympathetic control needs further investigations in females in relation to age and ovarian hormonal status.

Interestingly, analysis of the frequency domain components of the HRV in this study showed a significant increase in LF and LF/HF ratio in overweight females when compared to the control group suggesting sympathetic over activity, as higher values of LF/HF previously indicated greater sympathetic modulation of HRV.¹⁷

This observation could be a point of strength in this study as it highlighted the risk to which overweight late adolescent females are exposed due to cardiac sympathetic over activity. On the contrary, the LF power was significantly decreased in obese late adolescent females compared to the control group. In addition, the LF/HF ratio was insignificantly different in the same group indicating decreased sympathetic response in obese subjects. Previous researchers raised controversy

regarding sympathetic modulation of HRV in obese subjects.

Previously, it was stated that in spite of increased plasma noradrenaline level in the obese subjects, yet there was decreased end organ sympathetic responsiveness.¹⁸ Another study observed that there was no difference in sympathetic function between normal and obese individuals.¹⁶ On the other hand, higher sympathetic modulation of HRV was observed in obese girls with higher central fat compared to those with lower central fat.⁵

Postprandial hyperinsulinemia was reported in obese adolescents.¹⁹ Hyperinsulinemia has been shown to stimulate sympathetic nervous system via the hypothalamus or indirectly via the baroreflex in response to insulin-mediated vasodilation.²⁰ Explaining the present findings in view of the previous researches, we can hypothesize that in the overweight group there is hyperinsulinemia-mediated sympathetic overactivity, while in obese group, the repeated episodes of transient insulin resistance may underlie the diminished cardiac sympathetic activity. This hypothesis may solve the conflict between the researchers who postulated sympathetic overactivity and those postulated hypoactivity in obese subjects.

No significant correlation was observed in this study between BMI or WHR and HRV indices, although previously positive association between central fat and decreased parasympathetic and increased sympathetic activity was stated in overweight and obese girls.⁵

Further researches are still needed to solve the conflicts regarding effects of overweight and obesity on cardiac autonomic control in different age groups in females and the possible association between altered HRV and obesity indices.

CONCLUSION

This study postulates an important hypothesis. During the course of gaining weight, sympathetic hyperactivity was observed in late adolescent females possibly as a negative feedback to increase energy expenditure and prevent the increase in the body fat mass. This sympathetic overactivity may contribute to the high risk of cardiac problems in the overweight late adolescent females. On the other hand when obesity is already developed,

prolonged sympathetic over activity would result in a blunted sympathetic response.

Although females normally display enhanced protective parasympathetic input into cardiac regulation, yet this study revealed a diminished protective vagal cardiac modulation in overweight and obese late adolescent females.

REFERENCES

1. Sanisoglu SY, Okenli C, Hasimi A, et al. Prevalence of metabolic syndrome related disorders in a large adult population in turkey. *BMJ Public Health* 2006;6:92-92.
2. Poirier P, Giles TD, Bray GA, et al. Obesity and cardiovascular disease: Pathophysiology, evaluation, and effect of Weight loss. *Circulation* 2006;113:898-918.
3. Ogden CL, Yanovski SZ, Carroll MD, et al. The epidemiology of obesity. *Gastroenterology* 2007;132:2087-2102.
4. Kamath MV, Fallen EL. Power spectral analysis of heart rate variability: A non invasive signature of cardiac autonomic function. *Crit Rev Biomed Eng* 1993;21:245-311.
5. Miranda LS, Alves AJ, Aires SV, et al. Central fat influences cardiac autonomic function in obese and overweight girls. *Pediatr Cardiol* 2011;32:924-928.
6. Gao YY, Lovejoy JC, Sparti A, et al. Autonomic activity assessed by heart rate spectral analysis varies with fat distribution in obese women. *Obes Res* 1996;4:55-63.
7. Robillard ME, Bellefeuille P, Comtois AS, et al. The metabolically healthy but obese postmenopausal woman presents a favorable heart rate variability profile. *Scand Cardiovasc J* 2011;45(5):316-320.
8. Thayer JF, Yamamoto SS, BrossChot JF. The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *Int J Cardiol* 2010;141(2):122-131.
9. Wyatt SB, Winters KP, Dubbert PM. Overweight and obesity: Prevalence, consequences, and causes of a growing public health problem. *Am J Med Sci.* 2006;331(4):166-174.
10. Laederach-Hofmann K, Mussgay L, Ruddel H. Autonomic cardiovascular regulation in obesity. *J Endocrinol* 2000;164:59-66.
11. Kleiger RE, Stein PK, Bosner MS, et al. Time domain measurements of heart rate variability. *Cardiol Clin* 1992;10:487-498.
12. Akselrod S, Gordon D, Ubel FA, et al. Power spectrum analysis of heart rate fluctuation: A quantitative probe of beat-to-beat cardiovascular control. *Science* 1981;213:220-222.
13. Pomeranz B, Macaulay RJ, et al. Assessment of autonomic function in humans by heart rate spectral analysis. *Am J Physiol* 1985;248:151-153.
14. Reland S, Ville N, Wong S, et al. Does the level of chronic physical activity alter heart rate variability in healthy older women. *Clin Sci* 2004;107:29-35.
15. La Rovere MT, Pinna GD, Maestri R, et al. Short term heart rate variability strongly predicts sudden cardiac death in chronic heart failure patients. *Circulation* 2003;107:565-570.
16. Rossi M, Marti G, Ricordi L, et al. Cardiac autonomic dysfunction in obese subjects. *Clin Sci* 1989;76:567-572.
17. 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;93:1043-1065.
18. Piccirillo G, Vetta F, Fimognari FL, et al. Power spectral analysis of heart rate variability in obese subjects: Evidence of decreased Cardiac Sympathetic responsiveness. *Int J Obesity Relat Metab Disord* 1996;20:852-859.
19. Mager DR, Mazurak V, Rodriguez-Dimitrescu C, et al. A meal high in saturated fat evokes postprandial dyslipemia, hyperinsulinemia, and altered lipoprotein expression in obese children with and without nonalcoholic fatty liver disease. *J Parenter Enteral Nutr* 2013;37:517-28.
20. Gouloupoulou S, Baynard T, Franklin RM, et al. Exercise training improves cardiovascular autonomic modulation in response to glucose ingestion in obese adults with and without type 2 diabetes mellitus *Metabol Clin Exp* 2010;59(6):901-910.