

Obesity Impact on Heart Rate Variability Indices and the Modulation Effect of Regular Physical Activity

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Summary:

Background: Obesity is imposing a growing threat to world health. The autonomic nervous system (ANS) regulates visceral functions via balance between sympathetic and parasympathetic divisions. In the cardiovascular system (CVS) this non stationary balance results in the fluctuation between intervals of consecutive heart beats, so called heart rate variability (HRV). Obesity is one of the causative co-morbid conditions leading to metabolic and cardiac disorders as it is accompanied with varied combinations of abnormalities in the ANS, one view is that obese people have higher sympathetic tone. HRV measures the effect of autonomic function on the heart alone. Therefore, it could be the most useful method to investigate the effect of obesity on CVS as obesity is associated with decreased HRV. Regular physical activity has been shown to increase HRV in healthy individuals. Therefore, exercise training may improve cardiac autonomic regulation in a variety of clinical populations including obese individuals.

Objectives: to assess the alteration in cardiac autonomic function that may be associated with obesity by measuring HRV indices using (Holter monitoring) and to test the hypothesis that regular physical activity is associated with improved HRV.

Methods: A total of 49 asymptomatic obese individuals (28 males, 21 females) were recruited from Obesity Unit in Alkindy College of Medicine/ University of Baghdad.

Holter monitoring was applied to the obese individuals to assess HRV in addition to 47 healthy non-obese subjects of either sex as controls. All participants were subjected to 4-6 weeks moderate intensity of physical activity, and then Holter monitoring was repeated after the end of physical activity. Blood tests were done for all participants to exclude renal, liver and endocrine dysfunctions.

Results: all HRV indices were significantly low in obese subjects (p value less than 0.05) body mass index (BMI) was negatively correlated with overall HRV index ($p= 0.0001$, $r= - 0.87$). After onset of regular physical activity, all HRV indices were increased significantly in obese subjects whereas the increase in HRV indices in normal weight subjects did not reach the level of significance.

Conclusion: Obese subjects had a significant lower values of all HRV indices, overall low HRV values usually indicate a relative sympathetic dominance. BMI was negatively correlated with overall HRV index significantly in obese subjects. The significant increment in all HRV values in obese subjects after the onset of regular physical activity indicates a shift of sympathetic/parasympathetic balance towards increased vagal activity, which is a marker for cardiac autonomic modulation that may offset the negative effect of obesity.

Keywords: obesity, heart rate variability, physical activity, Holter monitoring, cardiac autonomic function.

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Introduction:

Obesity and weight gain are imposing a growing threat to world health, as in many countries 20%-30% of adults are categorized as clinically obese, and their number is still increasing.(1) The autonomic nervous system (ANS) regulates visceral functions through sympathetic and parasympathetic branches which act antagonistically to preserve dynamic equilibrium of vital functions. In the cardiovascular system (CVS) this non stationary balance results in the fluctuation between intervals of consecutive heart beats, so called heart rate HRV is a non-invasive, practical and reproducible measure

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of cardiac autonomic activity. HRV is the beat to beat variation in time of consecutive heartbeats under the balanced influence of sympathetic and parasympathetic components of the cardiac ANS expressed in normal sinus rhythm on electrocardiogram recordings, ranging from few minutes to 24-hours. (3) (4).

Obesity is one of the causative co-morbid conditions leading to metabolic and cardiac disorders as it is accompanied with varied combinations of abnormalities in the ANS, one view is that obese people have higher sympathetic tone. (5)

HRV also indicates the extent of neuronal damage to ANS. (6) It has also been suggested that a reduction in autonomic function might be the mechanism for increased prevalence of CVS disease in obesity.(7) However, these studies have not concentrated on the autonomic activity of the heart itself. HRV measures the effect of autonomic function on the heart alone.

Therefore, it could be the most useful method to investigate the effect of obesity on CVS disease. It is important to emphasize the effect of obesity on HRV, decreased HRV significantly increased CVS mortality (8), as obesity is related to increased morbidity and mortality in CVS disease (9). With the availability of new, digital, high frequency, 24-h multichannel electrocardiographic recorders, HRV has the potential to provide additional valuable insight into physiological and pathological conditions and to enhance risk stratification.(10). HRV can be assessed in two ways, either as Time Domain Analysis or in frequency domain as a Power Spectral Density (PSD) analysis. In either method, the time interval between each successive normal QRS complex are first determined. (11)

Regular physical activity has been shown to increase HRV in healthy individuals. (12) Therefore, exercise training may improve cardiac autonomic regulation in a variety of clinical populations including obese individuals. One hypothesis is that physical exercise modulates cardiac autonomic control by decreasing sympathetic influence and enhancing vagal tone. (13) There is also evidence that regular physical activity has a positive effect on HRV. (14)

Subjects and methods

A total of 49 asymptomatic obese individuals (28 males, 21 females) ranging in age from 25 to 60 years were recruited from Obesity Unit in Alkindy College of Medicine/ University of Baghdad, none of them had any history of hypertension, diabetes, cardiovascular disease, or stroke and/or taking any medications for these clinical conditions or any medication might affect the autonomic nervous system. Results of liver, renal, and endocrine function tests were all normal. In addition, 47 healthy non-obese subjects of either sex as controls. Anthropometric parameters like height and weight were recorded. Body mass index (BMI) was calculated as the weight in kilograms divided by the square of the height in meters. Obese were distinguished according to the WHO definitions (World Health Organization 1999): normal weight (18.5-24.9 Kg/m²), overweight (25-29.9 Kg/m²) and obese (≥30 Kg/m²).

HRV indices were measured for each subjects by using Holter digital device (DONGJIANG model: DJ-12A, CMICS Medical Instrument Co., Ltd, China), a 12-ECG leads were recorded over 24 Hour.

HRV was analyzed using the time domain method as it considered the original and simplest method for deriving HRV; time domain measures plot HRV as the change in normal R wave to R wave (RR) intervals over time.(15) Accordingly, HRV was computed for each subjects using the four standard 24-hour time domain measures: SDNN (standard deviation of all normal sinus RR intervals during a 24 hour period) which is the square root of their variance. A variance is mathematically equivalent to the total power of spectral analysis, so it reflects all cyclic components of the variability in recorded series of RR intervals, SDANN (standard deviation of the averages

normal sinus RR intervals in all 5-minute segments of the entire recording), rMSSD (the square root of the mean of the sum of the squares of differences between adjacent RR intervals), pNN50 (percentage of pairs of adjacent RR intervals differing by more than 50 millisecond (ms) in the entire recording). All the HRV indices with the exception of pNN50 are reported in units of time (ms).(16)

Time domain analysis was done according to the recommendations of Task Force of the European Society of Cardiology (ESC) and the North American Society of Pacing and Electrophysiology (NASPE). (11)

A 30-minute brisk walking three times per week program (moderate intensity) was applied to all participants for 4-6 weeks duration, HRV were assessed before and after physical activity.

Results:

Anthropometric characteristics of study subjects were summarized in table 1 and 2 from the table there was no significant differences in mean age and male/female ration between obese and control groups (p= 0.094, 0.99 respectively), while they differs significantly in BMI (p= 0.0001).

In table 3, HRV indices for obese and control groups were illustrated. From the table, obese subjects had a significantly lower values in the four studied HRV indices (SDNN, SDANN, rMSSD, and pNN50) with p value = 0.0001, 0.003, 0.0001 and 0.003 respectively as compared with control subjects.

Table 4 summarized the HRV indices before and after a period of 30 minutes brisk walking three times weekly for 4-6 weeks for both study groups. From the table, all HRV indices are increased after physical activity in both study groups (obese and control), however, obese had significant higher HRV indices after physical activity (p <0.05 in all HRV indices) as compared with HRV indices before the onset of the physical activity, while the increase in HRV indices in control subjects did not reach the level of significance (p > 0.05).

Table 5 showed a significant negative correlation between BMI and SDNN in obese subjects (p=0.0001, correlation coefficient, r= - 0.87), while in control subjects; there was negative correlation between BMI and SDNN but this correlation did not reach the level of significance (p= 0.095, r= - 0.42).

Table1: Anthropometric data of studied groups
BMI= body mass index

| Parameter | Obese n=49 | Control n=47 | P value |
|-------------------------------------|---------------|-----------------|---------|
| Age, mean ± SD (years) | 40.40 ± 6.45 | 37.98 ± 7.84 | 0.094 |
| BMI, mean ± SD (Kg/m ²) | 33.07 ± 2.58 | 22.88 ± 2.43 | 0.0001 |

Table 2: comparison between studied groups

| Parameters | Obese n=49 | | Control n=47 | | P value |
|------------|---------------|------|-----------------|-------|---------|
| | Frequency | % | Frequency | % | |
| Male | 28 | 57.2 | 27 | 57.44 | 0.99 |
| Female | 21 | 42.8 | 20 | 42.56 | |

Table 3: Comparison of heart rate variability (HRV) indices between obese and control subjects

| HRV indices | Obese | Control | P value |
|-----------------|--------------|---------------|---------|
| SDNN ± SD (ms) | 112.9 ± 26.1 | 161.8 ± 49.6 | 0.0001 |
| SDANN ± SD (ms) | 101.2 ± 40.3 | 137.01 ± 68.4 | 0.003 |
| rMSSD ± SD (ms) | 44.4 ± 19.2 | 74.5 ± 47.6 | 0.0001 |
| pNN50 ± SD (%) | 11.98 ± 8.71 | 17.28 ± 8.84 | 0.003 |

Table 4: HRV indices before and after physical activity in obese and control subjects

| HRV indices | Obese | | P value | control | | P value |
|----------------|--------------|---------------|---------|---------------|--------------|---------|
| | before | after | | before | after | |
| SDNN ± SD (ms) | 112.9 ± 26.1 | 142.7 ± 25.6 | 0.0001 | 161.8 ± 49.6 | 162.8 ± 25.9 | 0.9 |
| SDANN ± SD(ms) | 101.2 ± 40.3 | 127.97 ± 29.9 | 0.001 | 137.01 ± 68.4 | 151.9 ± 37.4 | 0.19 |
| rMSSD ± SD(ms) | 44.4 ± 19.2 | 63.8 ± 24.9 | 0.0001 | 74.5 ± 47.6 | 82.7 ± 40.0 | 0.37 |
| pNN50 ± SD (%) | 11.98 ± 8.71 | 15.96 ± 9.25 | 0.04 | 17.28 ± 8.84 | 17.77 ± 9.64 | 0.79 |

Table 5: Correlation between SDNN and BMI in obese and control subjects

| BMI (obese) | | |
|---------------|--------|---------|
| p-value | r | P |
| SDNN | - 0.87 | 0.0001 |
| BMI (control) | | |
| p-value | r | p-value |
| SDNN | - 0.42 | SDNN |

Statistics: Variables were expressed as Mean ± SD. Student's t test (unpaired 2 sample test) was used for comparing variables between study groups. Value of P < 0.05 was considered significant. Data were analyzed using MINITAB Release 16.1 of MINITAB statistical software.

Discussion:

The present study was designed to assess the effect of obesity on HRV which is one of the tools to evaluate the cardiac autonomic activity. In addition to assess the effect of regular physical activity on 49 obese and 47 normal weight subjects. HRV analysis is based on the concept that rapid fluctuations in heart rate may specifically reflect changes of sympathetic and vagal activity. Previous work has shown that pNN50 and rMSSD were measures of parasympathetic activity whereas SDNN and SDANN reflect both sympathetic and parasympathetic modulation of the heart rate (overall variability). (17)

HRV= heart rate variability; SDNN= standard deviation of all normal sinus RR intervals during a 24 hour period; SDANN= standard deviation of the averages normal sinus RR intervals in all 5-minute segments of the entire recording; rMSSD= the square root of the mean of the sum of the squares of differences between adjacent RR intervals; pNN 50= percentage of pairs of adjacent RR intervals differing by more than 50 millisecond.

This study showed that obese subjects had a significant reduction in all HRV indices as compared with control subjects. Overall low HRV values usually indicate a relative sympathetic dominance, which may be due to high sympathetic activity and/or low parasympathetic activity. (2) Decreased HRV indices in obese subjects was previously reported by Takayuki K. et al (18) and similar findings were reported by Facchini M. et al. (19) Other studies showed a significant reduction in parasympathetic activity with increasing body weight, which were similar to the present study. (20) (11)

BMI was negatively correlated with SDNN which is a measure of overall variability in both obese and normal weight subjects, although the correlation in obese subjects was significant. Consistent findings were reported by Quiliot D. et al and Maite V. et al. (21) (22) Lower HRV indices in obese subjects generally considered as an indicator of poorer autonomic function. lower HRV or autonomic dysfunction has been associated with several conditions of significant public health concern, including diabetes and diseases of the cardiovascular system.(23) Obesity, an important risk factor for diabetes and cardiovascular disease, is associated with dysregulation of autonomic function. It has been suggested that ANS dysfunction is an important mediator in the development of obesity-associated disease and insulin resistance although the nature of the link between obesity and insulin sensitivity is still unclear.(24) (25)

Furthermore, both study groups were subjected to 30 minutes

brisk walking three times weekly for 4-6 weeks (regular physical activity), the present study showed a significant increase in all HRV indices in obese subjects after the onset of physical activity, whereas HRV indices were also increased in control subjects although the increase in HRV did not reach the level of significance. High HRV values after the onset of regular physical activity indicate a shift of sympathetic/parasympathetic balance towards increased vagal activity. Similar findings were reported by Sui X. et al (26) as the improvement in HRV associated with regular physical activity for obese subjects. Thus the present data suggests that physical activity improves autonomic function as measured by HRV. These findings were analogous to findings reported by Denise FD. et al. (27) The underlying mechanisms by which regular physical activity improves vagal modulation are speculative at present, angiotensin II and nitric oxide (NO) are potential mediator. (28)

Conclusion:

Obese subjects had a significant lower values of all HRV indices, overall low HRV values usually indicate a relative sympathetic dominance, which may be due to high sympathetic activity and/or low parasympathetic activity. BMI was negatively correlated with overall HRV index significantly in obese subjects. High HRV values in obese subjects after the onset of regular physical activity indicate a shift of sympathetic/parasympathetic balance towards increased vagal activity which is a marker for cardiac autonomic modulation that may offset the negative effect of obesity.

Author s contributions:

Study conception: Dr. hayder sabah hassan, Dr.zaid almadfai, Dr. faris abdukkareem

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References:

1. Global Health Observatory Data Repository, world health organization 2008.
2. Borejda X, Olivia M, Massimiliano M, Carmine P, Rafffaele B. Heart Rate Variability Today. *Prog Cardiovasc Dis* 2012; 5:321-331. (IVSL)
3. McMillan DE. Interpreting heart rate variability sleep/wake patterns in cardiac patients. *J Cardiovasc Nurs* 2002; 17:69-81.
4. Freeman JV, Dewey FE, Hadley DM, Myers J, Froelicher VF. Autonomic Nervous System interaction with cardiovascular system during exercise. *Prog Cardiovasc Dis* 2006; 48: 342-62.

5. Jeong A, Yong-Gyu P, Kyung-Hwan C, Myung-Ho H, Hee-Chul H, Youn-Seon C, and Dokyung Y. Heart Rate Variability and Obesity Indices: Emphasis on the Response to Noise and Standing. *J Am Board Fam Pract* 2005; 18:97-103.
6. Chethan HA, Niranjana M, Basavaraju K. Comparative study of heart rate variability in normal and obese young adult males. *Int J Biol Med Res.* 2012; 3 (2): 1621-1623. (IVSL)
7. Laederach-Hofmann K, Mussgay L, Ruddle H. Autonomic cardiovascular regulation in obesity. *J Endocrinol* 2000;164:59-66.
8. Huikuri HV, Makikallio TH, Peng CK, Goldberger AL, Hintze U, Moller M. Fractal correlation properties of R-R interval dynamics and mortality in patients with depressed left ventricular function after an acute myocardial infarction. *Circulation* (2000) 101:47-53.
9. Vega GL. Results of Expert Meetings: Obesity and Cardiovascular Disease. Obesity, the metabolic syndrome, and cardiovascular disease. *Am Heart J* (2001) 142:1108-16.
10. Hohnloser SH, Kuck KH, Dorian P, et al. DINAMIT Investigators. Prophylactic use of an implantable cardioverterdefibrillator after acute myocardial infarction. *N Engl J Med* (2004) 351:2481-2488.
11. 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: 354-381.
12. Pichot V, Roche F, Denis C, et al. Interval training in elderly men increases both heart rate variability and baroreflex activity. *Clin Auton Res* (2005)15:107-15.
13. Malfatto G, Facchini M, Sala L, Branzi G, Bragato R, Leonetti G. Effects of cardiac rehabilitation and beta-blocker therapy on heart rate variability after first acute myocardial infarction. *Am J Cardiol* 1998;81:834-40.
14. Hottenrott K, Hoos O, Esperer HD. Heart rate variability and physical exercise. *Herz* (2006) 31(6):544-552.
15. Achten J, Jeukendrup AE. Heart rate monitoring: Applications and limitations. *Sports Med* 2003;33:517-38.
16. Axel Schäfer, Jan Vagedes. How accurate is pulse rate variability as an estimate of heart rate variability? A review on studies comparing photoplethysmographic technology with an electrocardiogram. *International Journal of Cardiology* (2013) 166; 15-29. (IVSL)
17. Li X, Shaffer ML, Rodriguez-Colon S, et al. The circadian pattern of cardiac autonomic modulation in a middle-aged population. *Clin Auton Res.* 2011;21:143-150.
18. Takayuki K, Noriko N, Yasushi H, Yoshika K, Hideki I, Toshio K, Tetsuya K, Michinori K. Effects of obesity, current smoking status, and alcohol consumption on heart rate variability in male white-collar workers. *Int Arch Occup Environ Health* (1997) 69: 447-454.
19. Facchini M, Malfatto G, Sala L, Silvestri G, Fontana P, Lafortuna C et al. changes of autonomic cardiac profile after a 3-week integrated body weight reduction program in severely obese patients. *J Endocrinol Invest* 2003 Feb;26(2):138-42.

20. Poirier P, Hernandez TL, Weil KM, Shepard TJ, Eckel RH. Impact of diet-induced weight loss on the cardiac autonomic nervous system in severe obesity. *Obes Res* 2003 Sep; 11(9):1040-7.
21. 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) 11:79–86.
22. Maite V, Manlio F, Marquez, Victor H, Borja-Aburto, Manuel Cardenas Antonio G, Hermosillo. Age, body mass index, and menstrual cycle influence young women's heart rate variability. *Clin Auton Res* (2005) 15 : 292–298.
23. H. Evrengul, H. Tanriverdi, S. Kose et al., "The relationship between heart rate recovery and heart rate variability in coronary artery disease," *Annals of Noninvasive Electrocardiology* (2006) vol. 11, no. 2, pp. 154–162.
24. S. Lindmark, L. Lönner, U. Wiklund, M. Tufvesson, T. Olsson, and J. W. Eriksson. "Dysregulation of the autonomic nervous system can be a link between visceral adiposity and insulin resistance," *Obesity Research* (2005) vol. 13, no. 4, pp. 717–728.
25. B. Gwen Windham, Stefano Fumagalli, Alessandro Ble, John J. Sollers, Julian F. Thayer, Samar S. Najjar, Michael E. Griswold, and Luigi Ferrucci. *The Relationship between Heart Rate Variability and Adiposity Differs for Central and Overall Adiposity*. Hindawi Publishing Corporation *Journal of Obesity* Volume 2012, Article ID 149516. (IVSL)
26. Sui X, LaMonte MJ, Laditka JN, Hardin JW, Chase N, Hooker SP et al. Cardiorespiratory fitness and adiposity as mortality predictors in older adults. *JAMA* (2007) 298(21):2507–2516.
27. Denise F D, Ursula AL, Christian S, Jean-Claude B et al. Effect of physical activity on heart rate variability in normal weight, overweight and obese subjects: results from the SAPALDIA study. *Eur J Appl Physiol* (2008) 104:557–565.
28. FS Routledge, TS Campbell, JA McFetridge-Durdle, SL Bacon. Improvements in heart rate variability with exercise therapy. *Can J Cardiol* 2010;26(6):303-312.