Research Article

# A cross-sectional study on effect of body mass index on the spectral analysis of heart rate variability

Umme Kulsoom Sheema, Basvaraj S Malipatil

Department of Physiology, M.R. Medical College, Kalaburgi, Karnataka, India.

Correspondence to: Umme Kulsoom Sheema, E-mail: ummekulsoom22@gmail.com

Received January 23, 2015. Accepted March 7, 2015

## Abstract

**Background:** Obesity is associated with dysregulation of autonomic function. Heart rate variability (HRV) in women has been related independently to menstrual cycle, body mass index (BMI), and other factors. A reduced HRV is associated with autonomic dysregulation. **Aims and Objectives:** The study was undertaken to observe the effect of BMI on the analysis of HRV in young adult females. **Materials and Methods:** A total of 60 healthy women aged 18–30 years participated in the study. Their BMI was measured after recording their height and weight using Quetelet's index. The HRV recording of each subject was then done. Using appropriate statistical tests, the HRV was compared among the subjects of different BMI groups. **Result:** HRV is reduced in overweight females depicted by an increase in low frequency (LF) and low frequency/high frequency ratio in overweight females when compared to normal-weight females. **Conclusion:** Raised BMI is associated with reduced HRV correlates with sympathetic overactivity and sympathovagal imbalance thereby increasing the chances of cardiac autonomic dysfunction, ultimately leading to cardiovascular disease in females.

**KEY WORDS:** heart rate variability; BMI; autonomic dysregulation

## Introduction

Heart rate variability (HRV) is reflective of the general wellness state of the organism. It is generally studied as the variation over time of the period between consecutive heart beats, and is predominantly dependent on the extrinsic regulation of the heart rate. HRV is thought to reflect the heart's ability to adapt to changing circumstances by detecting and quickly responding to unpredictable stimuli. HRV has become a popular method to study the state of the autonomic nervous system (ANS) regulating cardiac activity, and to study the physiological mechanisms responsible for the control of heart rate (HR) fluctuations, congestive heart failure, myocardial infarction, and other cardiac and noncardiac diseases.[1]

HRV in women has been related independently to endogenous sex hormones, hormone replacement therapy (HRT), menopause, menstrual cycle, BMI, and physical conditioning. With the aid of digital computers, it has become possible to study beat-to-beat HRV obtained from the R-R intervals in the ECG recordings. This method has proved to be of great clinical usefulness to evaluate the balance of sympathetic and parasympathetic regulation in several pathological conditions. A reduced HRV is associated with autonomic dysregulation.[2]

Obesity, an important risk factor for diabetes and cardiovascular disease, is associated with dysregulation of autonomic function.[3] Studies of the relationship between BMI, an indicator of obesity, and HRV have reported conflicting results with stronger associations observed in younger populations.[4] Understanding the mechanism that connects obesity and ANS function is important because of the increasing obesity prevalence documented among men and women of all ages, and low HRV is associated with sudden death.[5]

| Access this article online | |
|---|---|
| **Website:** http://www.njppp.com | Quick Response Code: |
| DOI: 10.5455/njppp.2015.5.2301201532 | |

HRV is a better marker of ANS than HR per se as the minimal changes in the ANS activity do not alter HR significantly but can be detected by HRV.[6] Spectral analysis of HRV is characterized by the high-frequency (HF) component (0.15–0.40 Hz) and low-frequency (LF) component (0.04–0.1 Hz). The HF component measures the influence of the vagus nerve in modulating the sinoatrial node, whereas the LF component provides an index of sympathetic effects on the heart, and the LF/HF ratio indicates sympathovagal balance. Decreased HRV reflects the increased sympathetic tone or decreased parasympathetic activity and is considered an important cardiovascular risk factor.[7]

The study was thus undertaken to observe the effect of BMI on the analysis of HRV in young adult females.

## Materials and Methods

Female medical students and female staff members of M.R. Medical College, Kalaburgi, were invited for the study among which 30 females with BMI of more than 25 kg/m$^2$ fulfilled the required criteria, whereas subjects with known respiratory, cardiovascular illness, or any disorder that could interfere the autonomic responses were excluded. These subjects formed the study group, whereas controls were 30 age-matched females with a BMI of the range 18.5–24.99 kg/m$^2$ from the same institute. Informed written consent was obtained from each subject after explaining the procedure. A detailed clinical history, including sociodemographic data, name, age, and occupation, was taken. A complete general physical examination and a systemic examination were conducted in all subjects. Subjects with known respiratory, cardiovascular illness, or any disorder that could interfere the autonomic responses were excluded.

### Anthropometric Variables

Anthropometric variables included in this study are as follows:

- Weight was recorded by using standard weighing machine whose least count was 0.5 kg.
- Height was measured by measuring scale whose least count was 0.1 cm. Height of each subject was converted into unit of meters.
- BMI was calculated by Quetelet's index as weight (in kg) divided by height in (meters)$^2$.
- BMI = weight (kg)/height (m$^2$)

Subjects were then divided into two groups based on their BMI: those with normal BMI 18.5–24.99 kg/m$^2$ in Group 1 (normal weight) comprising 30 females and those with BMI $>25$ kg/m$^2$ in Group 2 (overweight) comprising 30 females.

### Analysis of Heart Rate Variability

The HRV was recorded using ADInstruments PowerLab®/30 Series. The PowerLab is essentially a device specifically designed to perform the various functions needed for data acquisition, signal conditioning, and preprocessing. It contains its own microprocessor and specialized analog amplifiers for signal conditioning.

Subjects were instructed to remove any metallic objects such as watch or ornaments, and they were asked not to move their limbs during recording.

Skin surface was cleaned with alcohol swabs and then gel was applied to both wrists and right foot. Red electrode (–ve electrode) was placed over right wrist, black electrode (+ve electrode) was placed over left wrist, and green electrode (earth) was placed over right foot in supine position. These electrodes were connected to ADInstruments Bio Amp.

ECG recording was taken for 5 min in supine position with eyes closed and the HRV analysis for frequency domain (LF, HF, and LF/HF) was done. LF and HF spectral powers were determined by integrating the power spectrum between 0.04 and 0.15 Hz and 0.15 and 0.4 Hz, respectively. LabChart, version 8, was used for analysis and spectral powers were expressed in power percentage (%).

The values obtained were expressed as mean ± SD. Data were analyzed using statistical software SPSS, version 18. Unpaired student's $t$ test was applied to compare the significance of means between the two groups.

## Results

The basic variables are given in Table 1 showing the age, weight, height, and BMI of all the subjects.

Table 1 shows the anthropometric details of the study group comprising 60 young adult females with a mean age 21.085 ± 2.90 years, mean weight 55.96 ± 4.79 kg, and mean height 1.521 ± 0.06 m. The mean BMI was found to be 24.184 ± 3.531 kg/m$^2$.

Table 2 and show the relationship between HRV and BMI revealing that the females with BMI $>25$ kg/m$^2$ have an LF domain and LF/HF ratio greater than that of females with BMI $<25$ kg/m$^2$, the values being highly statistically significant ($p < 0.01$). The HF domain is higher in females with BMI $<25$ kg/m$^2$ than in females with BMI $>25$ kg/m$^2$, but the value is statistically non-significant ($p > 0.05$).

## Discussion

In this study, LF and LF/HF ratio was significantly increased ($p < 0.01$) in overweight females (BMI 25–29.9 kg/m$^2$) compared to normal-weight females (BMI 18.5–24.9 kg/m$^2$),

**Table 1:** Anthropometric details of study participants ($N$ = 60)

| Characteristics | Value (mean ± SD) |
|---|---|
| Age (years) | 21.085 ± 2.90 |
| Weight (kg) | 55.96 ± 4.79 |
| Height (m) | 1.521 ± 0.06 |
| BMI (kg/m$^2$) | |
| Overall | 24.184 ± 3.531 |
| Group 1 ($n$ = 30) | 20.964 ± 1.655 |
| Group 2 ($n$ = 30) | 27.40 ± 1.205 |

**Table 2:** Comparison of BMI with HRV

| HRV (%) | BMI (mean ± SD) | | *t*-Value | *p*-Value |
|---|---|---|---|---|
| | $<25$ kg/m$^2$ ($n$ = 30) | $>25$ kg/m$^2$ ($n$ = 30) | | |
| LF | 22.958 ± 6.32 | 58.43 ± 12.02 | 14.32 | $<0.0001$ |
| HF | 52.434 ± 14.7 | 45.98 ± 15.84 | 1.56 | $>0.05$ |
| LF/HF ratio | 0.4945 ± 0.250 | 1.442 ± 0.581 | 3.17 | $<0.01$ |

indicating sympathetic overactivity and sympathovagal imbalance, respectively. These results depict that HRV is reduced in overweight females. Gaining weight is associated with regular metabolic changes leading to "resistance" of feedback loops involved in organ systems, thereby causing diminished baroreceptor function.[8]

Increase in body fat may cause direct activation of sympathetic nervous system activity, increase in norepinephrine turnover, and decline in cholinergic activity at the cardiac level through leptin.[9]

Similar findings were observed in studies done by Krishna and Rao[10] and Soares-Miranda et al.,[11] where the LF nu was increased and HF nu was decreased in overweight individuals compared to normal-weight individuals.

Human obesity is characterized by marked sympathetic activation. A 10% increase in body weight above an individual's usual weight is accompanied with a decrease in parasympathetic activity. This effect of increased weight is one mechanism for cardiac alterations, such as arrhythmias, that accompany obesity.[12]

Some individuals seem to increase energy expenditure in response to overeating and, thus, maintain a stable body weight. The thermogenic adaptation that allows some individuals to resist weight gain despite overeating seems to be related to the activation of non-exercise activity thermogenesis (NEAT), which dissipates excess energy.[13] Interestingly, changes in ANS activity may play a role in the activation of NEAT .[14]

It is conceivable that the reduction in parasympathetic activity as body size increases may represent a defensive mechanism against fat deposition. However, only the concomitant increase in sympathetic activity may counteract the obesity-inducing effects of excess energy intake.[15]

## Conclusion

Raised BMI is associated with reduced HRV, which correlates with sympathetic overactivity and sympathovagal imbalance, thus increasing the chances of cardiac autonomic dysfunction and ultimately leading to cardiovascular disease in females.

## References


1. Stys A, Stys T. Current clinical applications of heart rate variability. Clin Cardiol. 1998;21:719–24.
2. Shetty SB, Pai SR. Comparison of cardiac autonomic activity and BMI in different phases of menstrual cycle using heart rate variability. Int J Basic Med Sci. 2011;2(4):100–5.
3. Liao D, Cai J, Barnes RW, Tyroler HA, Rautaharju P, Holme I, et al. Association of cardiac autonomic function and the development of hypertension: the Aric study Am J Hypertens. 1996;9(12I):1147–56.
4. Fagard RH, Pardaens K, Staessen JA. Influence of demographic, anthropometric and lifestyle characteristics on heart rate and its variability in the population. J Hypertens. 1999;17(11):1589–99.
5. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among us adults, 1999–2000. JAMA. 2002;288(14):1723–7.
6. Saul JP. Beat-to-beat variations of heart rate reflect modulation of cardiac autonomic outflow. News Physiol Sci. 1990;5:32–7.
7. 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;17:354–81.
8. Chaudhuri A, Borade NG. Menopause and autonomic control of heart. Med J DY Patil Univ. 2012;5:4–9.
9. Paolisso G, Daniela M, Montana N. Plasma leptin concentrations and cardiac autonomic nervous system in healthy subjects with different body weights. J Clin Endocrinol Metab. 2000;8(5):1810–4.
10. Krishna P, Rao D. Cardiac autonomic activity in overweight and underweight young adults. Indian J Physiol Pharmacol. 2013;57(2):146–52.
11. Soares-Miranda L, Alves AJ, Vale S, Aires L, Santos R, Oliveira J, et al. Central fat influences cardiac autonomic function in obese and overweight girls. Pediatr Cardiol. 2011;32:924–92.
12. Laederach-Hofmann K, Mussgay L, Ru H. Autonomic cardiovascular regulation in obesity. J Endocrinol. 2000;164:59–66.
13. Levine JA, Eberhardt NL, Jensen MD. Role of nonexercise activity thermogenesis in resistance to fat gain in humans. Science. 1999;283:212–4.
14. Vanltallie TB. Resistance to weight gain during overfeeding: a NEAT explanation. Nutr Rev. 2001;59:48–51.
15. Molfino A, Fiorentini A, Tubani L, Martuscelli M, Rossi Fanelli F, Laviano A. Body mass index is related to autonomic nervous system activity as measured by heart rate variability. Eur J Clin Nutr. 2009;63:1263–5.


**How to cite this article:** Sheema UK, Malipatil BS. A cross-sectional study on effect of body mass index on the spectral analysis of heart rate variability. Natl J Physiol Pharm Pharmacol 2015;5:250-252

**Source of Support:** Nil, **Conflict of Interest:** None declared.