

Relationship of body mass index to heart rate variability in young males.

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Abstract

Background: Heart rate variability (HRV) is one of the promising markers of cardiac autonomic activity. HRV is influenced by lifestyle related behaviours like physical inactivity and obesity.

Aim: To identify the relation between body mass index (BMI) and HRV in healthy young males.

Methods: 125 healthy male MBBS students in age group 17-20 yrs in BMI range 18-30kg/m² were selected for the study. BMI of subjects was calculated using formula BMI (kg/m²) = Weight(in kg) / [Height]²(in m²). HRV was recorded using frequency domain analysis method with AD INSTRUMENT POWERLAB®/30 SERIES. Ratio of low frequency (LF) component (0.04-0.15 Hz) and high frequency (HF) component (0.15- 0.4 Hz) i.e., LF/HF ratio which is an indicator of HRV was recorded. Relation between BMI and LF/HF ratio was analysed using Pearson's correlation test.

Results: A significant correlation of BMI with LF/HF ratio was observed (CC = 0.1971, p = 0.0276). LF/HF ratio was significantly high in those with BMI >25 kg/m² as compared to those with BMI <20kg/m² and BMI 20-25kg/m² (p < 0.05) using student's t test.

Conclusion: Decreased HRV reflects an abnormal autonomic sinus node activity characterized by a dominating sympathetic and reduced parasympathetic control. Autonomic nervous system (ANS) activity plays an important role in regulation of body weight and identifying subjects at risk for development of cardiovascular diseases.

Keywords: Heart rate variability, body mass index, autonomic nervous system, low frequency/high frequency ratio.

Introduction

Heart rate variability (HRV) has been established as a specific and sensitive non-invasive tool to study cardiac autonomic activity. HRV is an index of cyclical variations of beat-to-beat (RR) interval that reflects cardiac autonomic function and sympathovagal balance. It is also an indicator of the extent of neuronal damage to autonomic nervous system (ANS)^[1]. One of the methods for measuring HRV is the frequency domain method where low frequency (LF) parameter reflects primarily sympathetic modulation while high frequency (HF) reflects parasympathetic modulation. LF/HF ratio signifies efficiency of sympathovagal balance^[2]. Sympathovagal imbalance can explain increased

incidence of sudden cardiac deaths associated with obesity. Early intervention programs in young males through effective anti-obesity strategies can prevent cardiovascular morbidity and mortality in later ages. This study aims to correlate BMI, one of the parameters for obesity with HRV-related indices.

Methods

A total of 125 healthy MBBS male students in the age group of 17-20 years from a Medical College in Hyderabad were selected for the study. Only male subjects were selected to avoid gender differences which might influence results of the study. All the subjects were non-smokers, normotensive and euglycemic. None of the subjects were taking

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medications which affect ANS like β -blockers, calcium channel blockers, ACE inhibitors or angiotensin receptor antagonists. Body weight and height of subjects were measured and BMI was calculated using formula $BMI (kg/m^2) = \text{Weight}(\text{in kg}) / [\text{Height}]^2(\text{in } m^2)$. Subjects with BMI range 18-30 were included in the study. Subjects were further stratified according to their BMI into 3 groups: BMI $<20kg/m^2$, $20-25kg/m^2$ and $>25kg/m^2$. Informed written consent was taken from all the subjects. Clearance from Institutional Ethics Committee was taken. HRV was recorded using AD INSTRUMENT POWERLAB®/30 SERIES. Recording was taken in morning hours after having light breakfast. Each subject was made to rest for 5 minutes before taking the readings. The electrodes were connected to channel 3 of bioamplifier cable and lead II ECG was recorded for 5 min with the subject breathing normally. The ratio of two main frequency

components in frequency domain analysis i.e low frequency component(0.04-0.15 Hz) and high frequency component(0.15- 0.4 Hz) was recorded analysed^[3].

Results

Data was tabulated and analysed using Pearson's correlation test and student's t test using SPSS 17 software. Data was expressed as mean \pm S.D. p value <0.05 was considered statistically significant.

Mean body weight (in kg) of the subjects was 61.4 ± 8.80 and mean BMI was 21.43 ± 2.92 . LF/HF ratio was significantly high in those with BMI $>25 kg/m^2$ as compared to those with BMI $<20kg/m^2$ ($p = 0.0003$) and BMI $20-25kg/m^2$ ($p = 0.04$). LF/HF ratio of those with BMI $20-25kg/m^2$ was not significantly higher as compared to those with BMI $< 20kg/m^2$ ($p = 0.3$) (Table 1). BMI significantly correlated with LF/HF ratio ($CC = 0.1971$, $p = 0.0276$) {Table 2}.

Table 1. LF:HF ratio of study groups with varying BMI

Group	n	Weight	Height	BMI (kg/m ²)	LF:HF ratio
BMI<20	38	54.13 \pm 4.80	1.70 \pm 0.05	18.58 \pm 0.97	0.68 \pm 0.39
BMI 20-25	72	62.01 \pm 6.21	7.69 \pm 0.05	21.69 \pm 1.37	0.80 \pm 0.70
BMI >25	15	78.86 \pm 5.29	1.68 \pm 0.03	27.35 \pm 1.84	1.19 \pm 0.49

Table 2. Correlation between BMI and LF:HF ratio

HRV parameter	BMI	
	LF:HF ratio	CC
	P value	0.0276

CC – correlation coefficient.

Discussion

The present study was designed to assess effect of BMI on cardiac autonomic activity in healthy young males. Findings of study indicate that there is a positive correlation of BMI with LF/HF ratio which were similar to other studies^[4,5]. Increasing BMI is correlated to increased sympathetic and lower parasympathetic activities. A reduced HRV is a powerful and independent predictor of an adverse prognosis not only in patients with heart disease but also in the general population. Decreased HRV represents decreased vagal and increased sympathetic outflow to heart. It has been proven to be linked with a higher risk for hypertension, which

in turn leads to coronary heart disease and other cardiovascular diseases^[6].

Studies have revealed that ANS plays a central role in modifying energy expenditure and body fat content. Excessive storage of energy may be promoted by decreasing sympathetic activity and fat deposition is enhanced via increased parasympathetic activity. Thus, decreased parasympathetic activity as body size increases may represent defensive mechanism against fat deposition^[2,7]. This supports the view that activity of ANS influences body weight not only in obese but also healthy individuals.

A study conducted by Arrone L. et al^[8] demonstrated that 10% body weight gain reduces parasympathetic activity in non-obese while 10% weight loss increased parasympathetic and decreased sympathetic nervous system activity in both obese and non-obese^[9]. Lansberg et al^[10] hypothesized that increase in sympathetic nervous system activity with increased weight serves homeostatic role of increased thermogenesis which further prevents weight gain by increasing energy expenditure. Also, a

diet induced increase in plasma insulin concentration was primary mechanism mediating weight gain induced sympathetic neural activation^[11]. Studies have revealed that individuals who gain even modest amounts of weight contributing to increase BMI may experience increases in sympathetic nervous system activity regardless of whether they become obese. If not treated, sustained sympathetic activity may lead to development of hypertension and other cardiovascular disorders^[12]. However, sympathovagal balance seems to be genetically determined and therefore, may explain inter-individual differences in susceptibility to gain body weight^[2]. Thus, identification of a relation between ANS activity and BMI independent of presence of obesity may provide valuable insights into physiological regulation of body weight. Observing the association between BMI and HRV in healthy persons suggests that ANS dysfunction is a disordered homeostatic mechanism can be detected early and at relatively young ages, therefore providing an opportunity for early intervention.

Conclusion

The use of autonomic profiles holds promise for classifying human obesity and identifying subjects at increased risk for various cardiovascular disorders. Lifestyle modifications implemented early in life can serve as a preventive measure for sudden cardiac deaths in later ages.

Limitation

A prospective study can be undertaken in the same subjects to know the effect of weight reduction on cardiac autonomic activity.

References

1. 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.
2. Molfino A, Fiorentini A, Tubani L. Body mass index is related to autonomic nervous system activity as measured by heart rate variability. *Eur J Clin Nutr* 2009; 63: 1263-1265.
3. Liu CC, Terry BJ, Kuo and Cheryl Yang CH. Effects of estrogen on gender-related autonomic differences in humans. *Am J Physiol Heart Circ Physiol*. 2003; 285: 2188-2193.
4. Chethan HA, Niranjana Murthy, 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.
5. Koenig J, Jarczok MN, Warth M. Body mass index is related to autonomic nervous system activity as measured by heart rate variability--a replication using short term measurements. *J Nutr Health Aging*. 2014 Mar; 18(3):300-2.
6. Roy A, Kundu D, Mandal T. A comparative study of heart rate variability

tests and lipid profile in healthy young adult males and females. *Niger J Clin Pract* 2013; Vol 16(4):424-428.

7. Hugh R. Peterson, Marylee Rothschild, Clarice R. Weinberg. *Body Fat and the Activity of the Autonomic Nervous System*. *N Engl J Med* 1988; 318:1077-1083.
8. Arrone L, Mackintosh R, Rosenbaum M. Cardiac autonomic nervous system activity in obese and never-obese young men. *Obes Res* 1997; 5: 354-9.
9. Mohamed F Lutfi, Mohamed Y Sukkar. Relationship of height, weight and body mass index to heart rate variability. *Sudan Med J*. 2011; 47(1):14-19.
10. Landsberg L. Diet, obesity and hypertension: a hypothesis involving insulin, the sympathetic nervous system, and adaptive thermogenesis. *QJM*. 1986; 61: 1081-1090.
11. Christopher L. Gentile, Jeb S. Orr, Brenda M. Davy. Modest weight gain is associated with sympathetic neural activation in non-obese humans. *Am J Physiol Regul Integr Comp Physiol*. 2007; 292: 1834-1838.
12. Masuo K, Mikami H, Ogihara T, Tuck ML. Weight gain-induced blood pressure elevation. *Hypertension*. 2000; 35: 1135-1140.

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