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RESEARCH ARTICLE

Frequency domain analysis of heart rate variability in young obese adults with parental history of hypertension during isotonic exercise: A comparative study

Sudha B Sreenivas, Rajalakshmi R, Vijaya Vageesh Y

Department of Physiology, JSS Medical College, Musuru, Karnataka, India

Correspondence to: Sudha B Sreenivas, E-mail: sreenivassudha@yahoo.co.in

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ABSTRACT

Background: Heart rate variability (HRV) is the beat-to-beat variation in time of consecutive heartbeats. It is one of the indicators of autonomic nervous system (ANS) function. The ANS control of the cardiovascular system in obesity and hypertension remains controversial. **Aims and Objectives:** The objectives of the study were to analyze the effects of isotonic exercise on frequency domain indicators of HRV in young obese adults with parental history of hypertension and compare the same with that of normal controls along with an assessment of their recovery. **Materials and Methods:** Normotensive subjects in the age group of 18–22 years were selected and classified into two groups, based on body mass index and parental history of hypertension. Electrocardiogram was recorded before exercise, immediately after exercise and during recovery using powerlab. **Result:** Statistically significant variations were observed when values of low frequency (LF), high frequency (HF), and LF/HF ratio were compared in all the three conditions. Obese students with positive history of parental hypertension reflected a sympathovagal imbalance that exaggerated during exercise with delayed recovery. **Conclusion:** This study reveals the importance of vagal inhibition during exercise. It has also enabled us to detect cardiovascular autonomic imbalance that is exaggerated during exercises in young obese subjects who are genetically susceptible and more prone to develop hypertension later in life.

KEY WORDS: Heart Rate Variability; Isotonic Exercise; Obesity and Parental History of Hypertension; Frequency Domain Indicators of Heart Rate Variability

INTRODUCTION

Heart rate variability (HRV) is a physiological phenomenon, which is the beat-to-beat variation in time of consecutive heartbeats. It is the degree of fluctuation in the length of the intervals between heartbeats.[1] Although heart rate may be reasonably stable, the time interval between two heartbeats can be considerably different. Regulation and modulation of heart rate and its oscillations are dependent on autonomic nervous system (ANS).[2] HRV is an index that provides a non-invasive, practical, and reproducible measure of ANS function. Power spectral analysis of HRV provides a quantitative marker of autonomic neural control of heart rate. ANS responds dynamically to environmental changes resulting in increased HRV and a healthy heart, whereas a reduction in HRV indicates its unresponsiveness to change.[3] Evidence suggests that reduced HRV has poor prognostic significance for individuals with unstable angina, myocardial infarction, and chronic heart failure.[4] Interventions that reduce sympathetic activity and/or increase

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parasympathetic activity have been shown to protect against lethal arrhythmias.[5]

Obesity, an important risk factor for cardiovascular disease, is characterized by hemodynamic and metabolic alterations and is associated with deregulation of autonomic function. The WHO data reports that obesity has become an epidemic problem with 300 million obese persons worldwide. The ANS control of the cardiovascular system in obesity remains controversial.

Hypertension, an asymptomatic silent killer, is a well-known precursor of atherosclerotic cardiac disorders. Studies about the relationship between body mass index (BMI) and HRV have reported conflicting results with stronger associations observed in younger populations having high blood pressure and obesity.[6] There is lack of information about changes in autonomic control of cardiac functions in obese young adults.

Every physical activity requires quick adjustments on cardiovascular system to maintain circulatory homeostasis. Vagal withdrawal and a concomitant sympathetic activation, potentially modify the heart rate during exercise.[7] Recently, it has been reported that cardiovascular autonomic responses to isotonic exercise in healthy young adults with parental history of hypertension show signs of sympathetic overactivity.[8]

There are very few studies regarding effects of exercise on HRV in obese Indian population. The present study aims to focus on the evidence linking obesity in young adults and discuss the impact of parental hypertension in modulating these potential mechanisms during exercise. Any ANS dysfunctions in these subjects with a genetic predisposition to hypertension may serve as an early marker of cardiovascular disease risk factor. The exercises done by these subjects may also favor early detection of subclinical changes in their cardiac function.

Objectives of the Study

1. To study the effects of isotonic exercise on HRV in young obese adults with parental history of hypertension.
2. To compare the same with that of young adults with normal BMI without parental history of hypertension.

MATERIALS AND METHODS

The present study was conducted on students in the age group of 18–22 years. Estimation of sample size was based on mean and standard deviation (SD) for parameters under study as reported by Latha *et al*. To detect a reduction or oblique increase of 20% in the mean at 5% level of significance and with 80% power, the minimum sample size was calculated to be 30 in each group.[9]

Normotensive, voluntary subjects with sinus rhythm, without any acute systemic illness, non-smokers, and non-alcoholics were selected for this study. Regular athletes and those with cardiorespiratory diseases were not considered. Our study was approved by the Institutional Ethical Committee of JSS Medical College Mysuru. Subjects were explained about the purpose of the study, the study protocol and their informed consent was obtained.

Selected students were requested to complete a questionnaire that included specific information regarding age, parental history of hypertension, cardiac or pulmonary diseases, tobacco and alcohol intake, and physical activity. Clinical examination was conducted on all subjects to rule out any systemic disorders. Parental history of essential hypertension was ascertained from the medical prescription of the parents who were hypertensive and the anti-hypertensive medication prescribed, along with the dosage was noted in the datasheet. Subjects were instructed to refrain from caffeinated beverages 12 hours before the experiment and to avoid strenuous physical activity from the previous evening. Selected subjects were classified into two groups, based on BMI and parental history of hypertension.

Group 1 consisted of subjects with normal BMI (18.5–22.9 Kg/m^2), without parental history of hypertension (control group).

Overweight and obese subjects (BMI >23 Kg/m^2), with positive parental history of hypertension formed Group 2 (study group).

This study was conducted in our human physiology laboratory between 3 and 5 PM. Subject's age, height (in cm), and weight (in kg) were recorded using an electronic weighing scale and a stadiometer, respectively. BMI was calculated as the weight in kilograms divided by the square of the height in centimeters. Basal HRV was recorded for 5 min in lead II in supine position after 10 min of rest with eyes closed and the subject breathing normally during the recording. The subjects were acquainted with the procedure by trial runs. The exercising speed was set to the limit of experiment (6 Km/h), and the subjects were instructed to run on the treadmill for 12 min which corresponds to submaximal exercise. The second recording was done immediately following exercise on treadmill. Postexercise recovery period began from the cessation of exercise and lasted for the next 10 min. The third recording was done after the recovery period to study the recovery pattern.

Electrocardiogram (ECG) was recorded using Power lab® 8/30, ML870 high-performance data acquisition system with Lab chart® 7.1 software for windows. Signal acquisition, storage, and processing were performed on computer. All analyzed RR time interval series exhibited low noise (rate of erroneous RR intervals below 5 %). Before the computation, RR time interval series was corrected for artifacts using adaptive filtering. The digitized ECG signals were analyzed on-line and simultaneously stored on removable hard disks for off-line verification. 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 frequency domain indices - Low frequency band variation - LF (in $msec^2$) High frequency band variation - HF (in $msec^2$) a LF/HF ratio.[10]

Statistical Analysis

Arithmetic mean and SD was worked out to assess the values in both the groups. Student's *t*-test was done to assess the significance of changes in the autonomic functions among the groups. Repeated measures ANOVA were used in this study due to its robustness against many alternative distributions.[11]

RESULTS

Physical characteristics of the study were compared between the two groups suggesting statistical significance in BMI [Table 1].

In our study, LF values were compared in all the three conditions, namely, basal, immediately after exercise, and recovery ($P = 0.022$*) between the two groups. When the basal LF values were compared, a steady though not a statistically significant increase was observed inGroup 2. The LF values immediately after exercise also suggested a similar trend. Statistically significant difference was observed between the two groups during recovery period Table 2.

There was a statistically significant variation in our study when HF values were compared in all the three conditions, namely, basal, immediately after exercise, and recovery. Our study results showed a steady fall of mean values of HF between Group 1 and Group 2 and these values reduced further during exercise (enhanced sympathetic activity) Table 3.

Comparison of LF/HF ratio in basal and recovery readings showed a statistical significance. This ratio demonstrated a significant increase during exercise with a slow recovery in Group 2, with a persistent increase in the ratio during recovery period which was statistically significant ($P = 0.001$*). Group 1 showed a recovery back to near normal basal values Table 4.

Table 1: Mean age and BMI of the groups

Groups	n=60	(mean±SD)	
		Age	BMI
Normal BMI without parental H/O hypertension	30	18.90±0.96	21.71±1.73
Obese with parental H/O hypertension	30	19.20±1.10	27.94±2.79
T		1.125	10.34
P		0.233	0.001*

*P value <0.05. BMI: Body mass index, SD: Standard deviation

Table 2: Mean and SD values of LF (in m/s^2) during basal, exercise, and recovery period between the groups

Conditions	Group 1	Group 2	T	*P* value
Basal	151.2±105.5	176.3±115.2	0.878	0.383
Immediately after exercise	216.6±174.9	224.7±95.5	0.222	0.824
Recovery	153.9±173.7	242.7±112.4	2.35	0.022*

*P value <0.05. SD: Standard deviation, LF: Low frequency

Table 3: Mean and SD values of HF during basal, exercise, and recovery period between the groups

Conditions	Group 1	Group 2	T	*P* value
Basal	306.2±203.1	212±103.9	2.261	0.0028*
Immediately after exercise	212.7±157.0	210.6±153.3	0.0524	0.95
Recovery	215±100.8	188.3±139.9	0.848	0.400

*P value <0.05. SD: Standard deviation, HF: High frequency

Table 4: Mean and SD values of LF/HF during basal, exercise, and recovery period between the groups

Conditions	Group 1	Group 2	T	*P* value
Basal	0.67±0.59	1.06±0.91	1.96	0.054
Immediately after exercise	1.18±0.70	2.05±2.57	1.789	0.082
Recovery	0.71±0.41	1.8±1.09	5.126	0.001*

*P value <0.05. SD: Standard deviation, HF: High frequency, LF: Low frequency

DISCUSSION

HRV is a valuable tool to evaluate the autonomic regulation of heart rate.[12] Sympathetic-parasympathetic interactions bring about significant differences in HRV complexity.[13,14] Power spectral analysis of HRV typically identifies two main frequency-domain components; the first is a LF component around 0.1 Hz and is predominantly considered a marker of sympathetic modulation of the cardiovascular system. The second is a HF component around 0.3 Hz that reflects parasympathetic modulation. In autonomic states between rest and light exercise, the actual heart rate is determined by simultaneous sympathetic acceleration and vagal deceleration.[15] When a person begins to exercise, there is parasympathetic withdrawal and sympathetic activation. On cessation of exercise, there is parasympathetic reactivation.

The changes in LF and HF powers and in LF/HF ratio observed soon after isotonic exercise reflects the decrease in vagal activity and the activation of sympathetic system. Our study specified a similar trend with increase in the LF values in both the groups immediately following isotonic exercise.

This study also revealed that the values of LF and LF/HF ratio following exercise were significantly higher in Group 2 that persisted even during the recovery period.

The basal HF values were comparatively reduced in Group 2 implying sympathetic dominance in obese individuals even at rest conditions. Group 1 showed a considerable fall in postexercise HF values with rapid recovery to basal levels indicating a very good control of parasympathetic tone. This feature was not observed in Group 2. Furthermore, there was a substantial reduction in HF power values in Group 2, the probable assertion being impaired vagal tone.

The LF-HF ratio of the study group was marginally higher than that of the control subjects, suggesting a deregulated sympathovagal balance in the study group.

The results of our study are consistent with other studies that state that LF not only corresponds to sympathetic activity but also provides an index of the baroreceptor reflex modulation of sympathetic activity.[16,17] One study showed the evidence of poor vagal rebound after exercise in the group with H/O parental hypertension.[18] Probably, a similar mechanism of poor parasympathetic reactivation was operating in our study group too. Studies have shown that normotensives with a family history of hypertension exhibit altered sympathovagal balance with decreased parasympathetic activity.[19]

Signs of altered responses to physical exertion were observed in obese individuals who have a remarkably poor ability to modify their ANS axis. Those with a parental history of hypertension also showed an exaggerated response to exercise testing.[20] Studies found that obese subjects had an elevated LF power values even at rest, probably reflecting imbalance in their autonomic vasomotor activity.[21] Our findings endorse this statement and elucidate that excess body weight has a vicious impact favoring abnormal sympathetic modulation of the cardiovascular system.

This study has highlighted an increase in all the spectral indices that reflect a predominant sympathetic modulation of the heart. An imbalance in the autonomic neural activities of the heart in obese subjects has been demonstrated.[22] This is featured in obese and hypertensive subjects suggesting a genetically determined sympathetic overactivity and an increased sympathetic drive to the vessels. There is considerable evidence to suggest that autonomic dysregulation is present in the early stage of hypertension.[23] Reports confirm that an exaggeration of increased sympathetic activity facilitates the onset of hypertension in prehypertensives.[24,25]

A noteworthy feature in our study was that the LF component of HRV in the study group was elevated in all the three conditions. This finding confirms the compounded adverse effects of obesity and parental hypertension and the susceptible individuals show a strong sympathetic overactivity, which may expose them to cardiovascular risks in future.

It is vital to understand the mechanisms that drive the altered hemodynamic responses during exercise in obese individuals to prevent the progression to hypertension. In spite of limitation of less sample size and wide distribution of values, the present study emphasizes the necessity to improve vagal tone and lower sympathetic tone in obese young adults with hypertensive parents, so that they do not progress into the stage of prehypertension.[26]

CONCLUSION

This study reveals the importance of vagal inhibition during exercise. It has also enabled us to detect cardiovascular autonomic imbalance that is exaggerated during exercises in young obese subjects who are genetically susceptible and more prone to develop hypertension later in life. Early interventions in the form of dietary and lifestyle modifications with regular exercise programs, relaxation techniques, etc., for obese subjects with hypertensive parents might delay or avoid the onset of hypertension and maintain their sympathovagal homeostasis.

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REFERENCES

1. Marek M, John CA. Measurement of heart rate variability. J Am Coll Cardiol 1995;25:433-7.
2. Williamson JW, Fadel PJ, Mitchell JH. New insights into central cardiovascular control during exercise in humans: A central command update. Exp Physiol 2006;91:51-8.
3. McMillan DE. Interpreting heart rate variability sleep/wake patterns in cardiac patients. J Cardiovasc Nurs 2002;17:69-81.
4. Sandercock GR, Paul DB, David AB. Effects of exercise on heart rate variability: Inferences from meta-analysis. Med Sci Sports Exerc 2005;37:433-9.
5. Kendall MJ, Lynch KP, Hjalmarson A, Kjekshus J. Beta-blockers and sudden cardiac death. Ann Intern Med 1995;123:358-67.
6. Windham BG, Fumagalli S, Ble A, Sollers JJ, Thayer JF, Najjar SS, *et al.* The relationship between heart rate variability and adiposity differs for central and overall adiposity. J Obes 2012;149516.
7. Nishiyasu T, Tan N, Morimoto K, Nishiyasu M, Yamaguchi Y, Murakami N, *et al.* Enhancement of parasympathetic cardiac activity during activation of muscle metaboreflex in humans. J Appl Physiol (1985) 1994;77:2778-83.
8. Sowmya R, Maruthy KN, Gupta R. Cardiovascular autonomic

responses to whole body isotonic exercise in normotensive healthy young adult males with parental history of hypertension. Indian J Physiol Pharmacol 2010;54:37-44.
9. Latha R, Rajalakshmi R, Ratchagan K. Assessment of cardiac sympatho vagal activity in overweight young adult males. Natl J Physiol Pharm Pharmacol 2016;6:101-5.
10. 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.
11. David SM, George P, McCabe B, Craig A. Introduction to the Practice of Statistics. 6th ed. New York: W.H. Freeman and Company; 2003. p. 763.
12. Goldberger JJ, Ahmed MW, Parker MA, Kadish AH. Dissociation of heart rate variability from parasympathetic tone. Am J Physiol 1994;266:H2152-7.
13. Lind AR, Mc Nicol GW. Muscular factors which determine the cardiovascular responses to sustained and rhythmic exercise. Can Med Assoc J 1967;96:706-15.
14. Matthias W, Kristin B, Annika R, Regina S, Steffi K. Heart rate variability and blood pressure during dynamic and static exercise at similar heart rate levels. PLoS One 2013;8:e83690.
15. Bootsma M, Wenne CA, Janssen MJ, Cats VM, Schalij MJ. Heart rate variability and sympatho vagal balance: Pharmacological validation. Neth Heart J 2003;11:250-8.
16. Perini R, Veicsteinas A. Heart rate variability and autonomic activity at rest and during. Exercise in various physiological conditions. Eur J Appl Physiol 2003;90:317-25.
17. Radaelli A, Bernardi L, Valle F, Leuzzi S, Salvucci F, Pedrotti L, *et al.* Cardiovascular autonomic modulation in essential hypertension. Effect of tilting. Hypertension 1994;24:556-63.
18. Mezzacappa ES, Kelsey RM, Katkin ES, Sloan RP. Vagal rebound and recovery from psychological stress. Psychosom Med 2001;63:650-7.
19. Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. Circulation 1991;84:482-92.
20. Wilson MF, Sung BH, Pin comb GA, Lovallo WR. Exaggerated pressure response to exercise in men at risk for systemic hypertension. Am J Cardiol 1990;66:731-4.
21. Grassi G, Serravalle G, Cattaneo BM, Bolla GB, LanfranchiA, Colombo M, *et al*. Sympathetic activation in obese normotensive subjects. Hypertension 1995;25:560-3.
22. Rajalakshmi R, Vageesh YV, Nataraj SM, Dhar M, Srinath CG. Heart rate variability in Indian obese young adults. Pak J Physiol 2012;8:39-44.
23. Singh JP, Larson MG, Tsuji H, Evans JC, O'Donnell CJ, Levy D. Reduced heart rate variability and new-onset hypertension insights into pathogenesis of hypertension: The Framingham heart study. Hypertension 1998;32:293-7.
24. Malliani A. Heart rate variability: From bench to bedside. Eur J intern Med 2005;16:12-20.
25. Pal GK, Adithan C, Amudharaj D, Dutta TK, Pal P, Nandan PG, *et al.* Assessment of sympathovagal imbalance by spectral analysis of heart rate variability in prehypertensive and hypertensive patients in Indian population. Clin Exp Hypertens 2011;33:478-83.
26. Dipla K, Zafeiridis A, Koidou I, Geladas N, Vrabas IS. Altered hemodynamic regulation and reflex control during exercise and recovery in obese boys. Am J Physiol Heart Circ Physiol 2010;299:H2090-6.

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