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Autonomic function in hypertensive patients and their relation to BMI

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Abstract

Heart Rate Variability (HRV) has emerged as a practical, non-invasive tool to quantitatively investigate cardiac autonomic dysregulation. Obesity, hypertension and diabetes mellitus are known to be associated with dysregulation of autonomic functions independently. The present study was undertaken to ascertain whether obesity has any effect on further disruption of autonomic functions particularly in hypertensive patients. HRV was determined in 32 obese and 32 non-obese age and sex matched hypertensive patients. One minute HRV was analyzed during deep breathing both in test group and control group. HRV was significantly decreased in obese hypertensive patients than non-obese hypertensive patients. Our present study supports that obesity and hypertension probably has additive effect in causing autonomic dysregulation.

Keywords: Autonomic functions, HRV, hypertension, obesity

Introduction

Autonomic nervous system (ANS) is the portion of nervous system that controls most of the visceral functions in the body. It controls various organs of the body through two major divisions called the sympathetic nervous system & the parasympathetic nervous system [1]. The two divisions produce antagonistic effects on different organs. Thus, ANS plays important role in maintaining Homeostasis/millieuinterieur. Dysregulation of autonomic functions is seen in obesity [2], hypertension [3], and diabetes mellitus [4]. HRV is a term used to describe variations in both instantaneous heart rate & R-R interval set [5]. There are regular fluctuations in heart rate, which are primarily due to changing level of both parasympathetic & sympathetic neural control of the heart [6, 7].

Beat to beat fluctuation in heart rate (HR) or variation in R-R interval are conventionally described with the term Heart rate variability (HRV). HRV is a non-invasive electrocardiographic marker reflecting the activity of sympathetic and vagal components of the ANS on sinus node of the heart. It expresses the total amount of variations of both instantaneous HR & RR intervals [8, 9] although HR changes are independent of HRV [10]. Thus HRV analyses the tonic baseline autonomic function. In normal heart with an intact ANS, there will be continuous physiological variations of sinus cycles reflecting a balanced sympathovagal state and normal HRV. In damaged heart which suffered necrosis, the change in activity of afferent & efferent fibers of the ANS & on local neural regulation will contribute to the resulting sympathovagal imbalance reflected by a diminished HRV.

HRV with deep breathing (HRVdb) is highly sensitive measure of cardiovagal or sympathetic cardiac function and defined as the difference in beats/minute between the shortest and the longest heart rate interval measured by electrocardiographic recording during six cycles of deep breathing.

Therefore, HRVdb is a reliable & sensitive clinical test for early detection of cardiovagal dysfunction in a wide range of autonomic disorders [11]. HRV reflects the balance between sympathetic and parasympathetic tone: when sympathetic tone is dominant HRV is low and vice-versa [9].

Obesity, hypertension and diabetes mellitus are known to be associated with dysregulation of autonomic functions independently. The present study is undertaken to ascertain whether obesity with hypertension has additive effect in further disruption of autonomic functions.

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Materials and Methods

Study was carried out at Physiology Dept between 2018-2019. Informed and written consent was taken from the subjects participating in the study. Our study did not involve any invasive procedure. Ethical clearance was taken from Institutional Ethical Clearance Committee.

Study included 32 obese hypertensives (Group I), 32 non-obese hypertensives (Group II) and 32 non-obese normotensive (control) of both sex, aged 40-50 years visiting the primary health centre.

Males & Females both having BMI of $\geq 25 \text{ kg/m}^2$ were considered obese, based on BMI cut off for Indian population. Average BP measuring $\geq 140/90$ mmHg in both males & females i.e. freshly diagnosed to have high BP after three different readings on different days were considered as hypertensive.

Average BP of $\leq 130/80$ mm Hg after three different readings in both genders were considered normotensive. Hypertensives who are on regular treatment, smokers and alcoholics were excluded from the study.

HRV was measured using a simple bedside test of 1 minute HRV during forced deep breathing. The test was performed using CARDIART 6108-T electrocardiograph. ECG was taken every morning between 10 to 11 am after abstinence of coffee, tea and Cola for 8 hrs by the patients. The subjects were made to lay down quietly in a supine posture and Lead II of the ECG machine was connected to record the heart rate. After obtaining a stable heart rate record, the patients were instructed to breathe deeply at 6-8 breaths/min (for one respiratory cycle, time taken was 10 secs, 5 sec inspirations and 5 sec expiration). Lead II was then recorded continuously at paper speed of 25 mm/sec for one minute. The HRV interval (R-R intervals between adjacent QRS complexes resulting from sinus node depolarisation) was measured manually with a scaled calliper. R-R intervals surrounding premature ventricular contractions were excluded.

One minute HRV was calculated as the difference in beats/minute between the shortest and the longest heart rate interval [4].

Results

In this study Independent sample *t*-test and correlation was employed for statistical comparison of HRV between the test groups and control group, using SPSS-16 for windows. The results are presented as Mean \pm SD. The $p < 0.05$ was considered statistically significant.

The average age of the two test and control groups was similar (40 – 50 yrs).

There was decrease in mean minimal Heart Rate and One minute HRV, which was observed during deep breathing among obese and non-obese hypertensives on comparison with that of healthy controls and it was statistically significant. Further, statistically significant decrease in mean minimal heart rate and One minute HRV was observed during deep breathing among obese hypertensives when compared with non-obese hypertensives.

There was no significant difference in mean maximal heart rate between obese hypertensive and non-obese hypertensives as well as non-obese hypertensive and control group, but significant difference was observed between obese hypertensive and control group.

The basal HR in obese and non-obese hypertensives were high compared to controls. There was no association between the HR and HRV in both obese and non-obese hypertensives.

Table 1: The characteristics of patients and controls

Parameters	Non-obese hypertensive Patients (Group I)	Obese hypertensive Patients (Group II)	Controls
Number	32	32	32
Male	17	17	17
Female	15	15	15
Age (yr)	54.21	54.84	53.40
HR	76 beats/min	80 beats/min	73 beats/min

Table 2: Comparison of maximum, and minimum HR, and HRV between group I and controls

Parameters	Non-obese hypertensive Patients (Mean \pm SD)	Control (Mean \pm SD)	t-value	p-value
Maximum HR	88.691 \pm 1.053	91.031 \pm 2.174	-3.137	.060
Minimum HR	69.686 \pm 1.761	66.251 \pm 2.082	2.973	.004*
HRV	19.005 \pm 1.639	24.757 \pm 2.101	-5.841	.000*

HR=Heart Rate, HRV=Heart Rate Variability, *highly significant

Table 3: Comparison of maximum, and minimum HR, and HRV between group II and control

Parameters	Obese hypertensive Patients (Mean \pm SD)	Control (Mean \pm SD)	t-value	p-value
Maximum HR	84.833 \pm 1.447	91.031 \pm 2.174	-6.926	0.046
Minimum HR	71.826 \pm 0.969	66.251 \pm 2.082	13.993	0.000*
HRV	13.007 \pm 1.465	24.757 \pm 2.101	-19.560	0.000*

HR=Heart Rate, HRV=Heart Rate Variability, *highly significant

Table 4: Comparison of maximum, and minimum HR, and HRV between group I and group II

Parameters	Obese hypertensive Patients (Mean \pm SD)	Non-obese hypertensive Patients (Mean \pm SD)	t-value	p-value
Maximum HR	84.833 \pm 1.447	88.691 \pm 1.053	-5.870	0.000*
Minimum HR	71.826 \pm 0.969	69.686 \pm 1.761	11.956	0.008*
HRV	13.007 \pm 1.465	19.005 \pm 1.639	-15.706	0.000*

Discussion

The arterial pressure of hypertensive and obese individuals is more labile to various forms of physical stress than those of normotensive and normal weight individuals, indicating altered baroreceptor reflex i.e. autonomic system which can be detected by HRV. This study was carried out to know the HRV response in newly diagnosed non-obese hypertensives and obese hypertensives along with controls to know the presence of additive effects of HRV variations in Obese hypertensive patients. If obesity can change the HRV response as well as hypertension, then obese hypertensive patients should have more HRV alterations which can be dangerous to their health. To investigate this hypothesis, HRV was measured and the result showed the reduction in HRV in obese & non-obese hypertensives compared with normal healthy individuals which is in line with previous population based study [11]. Also, reduction in HRV was observed more in obese hypertensives than non-obese hypertensives.

Prevalence of overweight, obesity as well as hypertension in India shows the increasing trend because of lifestyle changes among the population. Obesity is also on the rise with excessive consumption of processed foods and high fat diets. WHO had reported that prevalence of obesity is increasing

world wide including the developing countries [12]. Over weight and obesity itself are the independent risk factors for developing many diseases. Hypertension presents mainly because of genetic alterations and environmental effects. Both obesity and hypertension have higher sympathetic tone which is proved from elevated catecholamine levels [13]. When sympathetic stimulation is high it releases renin from the kidney which in turn increases the angiotensin levels. Angiotensin II has positive effect on sympathetic ganglion and adrenal medulla while the negative effect on vagal outflow to the heart [11] which will decrease the HRV.

The present study shows that HRV is uniformly reduced in newly diagnosed hypertensive patients. There is slightly altered max HR and gross variations in min HR between obese and non-obese hypertensives as well as non-obese hypertensives and controls in our study, thus indicating reduced sympathetic tone with near normal parasympathetic tone, which is in concordance with the study carried out by Virtanen. R and A Jula. A *et al.* [11] Not only the freshly diagnosed HTN was associated with decreased HRV but also the association between HRV and blood pressure was present across the full range of blood pressure which is similar to the study by Emily B. Schroeder and Duanping Liao *et al.* [14] Previous studies have shown reduced HRV in patients with hypertension alone [15] & in persons with obesity alone [2, 16]. Our study revealed a first of its findings that there is highly significant reduced HRV with gross variations in max HR and min HR between obese hypertensives and control indicating alteration in both parasympathetic and sympathetic outflow, which is evident by the more positive correlation of HRV with obese hypertensives ($r=0.98$) compared to non-obese hypertensives ($r=0.76$). High levels of angiotensin II may be the probable cause for the gross variations, putting these patients in high risk category for early development of coronary artery disease. Short term regulation of heart rate is predominantly by sympathetic & parasympathetic system, so autonomic imbalance can be known by heart rate fluctuations [2]. Our study shows that HR was higher in obese hypertensives when compared to non-obese hypertensives & control group but the association of blood pressure with HRV was independent of HR.

The study by Reaven *et al.*, palatine and julius *et al.* as well as Julius and Nesbitt *et al.* [14] have proven that autonomic nervous system dysregulation precedes the development of clinical hypertension and obesity independently. Decreased Parasympathetic activity in obesity was reported by Aronne *et al.* and Rossi *et al.* [17] Obesity is associated with impaired glucose tolerance and hyperinsulinemia contributing to low cardiac vagal activity. Thus obese individuals suffer from increased mortality risk due to cardiovascular disorder which gets worse if they develop Hypertension [2].

In this century we have amassed new knowledge about physical activity and associated positive implications on health. Almost all physiological process will be benefited by the regular physical activity. In fact, now a days it has taken first line of management in treating several chronic illnesses and also in rehabilitation. Different types of exercise have its own effects on the human body. A working party of the Royal College of Physicians, convened in 1989, examined this evidence, recognized its importance and based a series of recommendations on it [18]. So not only identifying the autonomic imbalance through HRV can reduce the risk of developing hypertension and obesity but also by early intervention of life style changes and exercise can have a

remarkable health improvement in Indians.

Conclusion

Regular monitoring of HRV and detection of autonomic instability and rehabilitative measures can not only reduce the incidence of hypertension and obesity but may be diabetes also where the prevalence has crossed >50% in Indian population. The limitation of this study was that duration of obesity was not estimated. Prospective studies are needed to find out whether reduced heart rate variability identifies hypertensive subjects with increased risk of cardiac mortality.

Conflict of interest: None declared.

References

1. Guyton & hall textbook of physiology 12 edition pp 729 publ- Elsevier Saunders.
2. Rajalakshmi R, Vijaya Vageesh Y, Nataraj SM, MuraliDhar, Srinath CG. heart rate variability in indian obese young adults. Pak J Physiol. 2012; 8(1):39-44.
3. Pandurang Narhare, Chaitra B, Surender T. A comparative study of cardiovascular autonomic function in hypertensive and normotensive people. Int. J Pharm Biomed Res. 2011; 2(4):223-226.
4. Fareedabanu AB, Gorkal AR, Narsimha Setty KR. A simple test of one-minute heart rate variability during deep breathing for evaluation of sympathovagal imbalance in patients with type 2 diabetes mellitus. Pak J Physiol. 2011; 7(1):33-36.
5. Task force of European society of cardiology. North American society of pacing & electrophysiology. Heart rate variability: standards of measurements physiological interpretation & clinical use. Circulation. 1996; 93:1043-65.
6. Nakamura Y, Yamamoto Y, Muraoko I. Autonomic control of heart rate during physical exercise & fractal dimension of heart variability. J Appl Physiol. 1993; 74(2):875-81.
7. Arai Y, Saul JP, Albrecht P *et al.* Modulation of cardiac autonomic activity during & immediately after exercise. Am J Physiol. 1989; 213:1322-41.
8. Stein PK, Bosner MS, Kleiger RE, Conger BM. Heart rate variability: A measure of cardiac autonomic tone. Am Heart J. 1994; 127:1376-81.
9. Van Ravenswaaij-Arts CMA, Kollee LAA, Hopman JCW, Stoelinger GBA, Van Geijn HP. Heart rate variability. Ann Intern Med. 1993; 118:436-47.
10. Virtanen R, Jula A, Kuusela T, Helenius H, Voipio-Pulkki LM. Reduced heart rate variability in hypertension: associations with lifestyle factors and plasma renin activity. Journal of Human Hypertension. 2003; 17:171-179.
11. Shields RW. Heart rate variability with deep breathing as a clinical test for cardiovagal function. Clin J Med. 2009; 76(2):37-40.
12. Archana Damodaran and Balasubramanian Kabali. Autonomic Dysfunction in Central Obesity. World Journal of Medical Sciences. 2013; 8(2):118-122.
13. Jeong A Kim *et al.* Heart Rate Variability and Obesity Indices: Emphasis on the Response to Noise and Standing. JABFP, 2005, 18(2).
14. Emily B Schroeder, Liao D, Chambless LE, Prineas RJ, Evans GW, Heiss G. Hypertension, Blood Pressure, and Heart Rate Variability: The Atherosclerosis Risk in Communities (ARIC) Study. Hypertension. 2003;

42(6):1106-1111.

15. Mohd.Urooj, Pillai KK, Monika Tandon, Venkateshan SP, Nilanjan Saha. Reference ranges for time domain parameters of heart rate variability in Indian population and validation in hypertensive subjects and smokers. *Int. J Pharm Pharm Sci.* 2011; 3(1):36-39.
16. Masari Amano, Tomokanda, Hidetoshi UE, Toshio moritani. Exercise training and autonomic nervous system activity in obese individuals. *Medicine & Science in Sports & Exercise*, 2000, 1287-1291.
17. Gwen Windham B *et al.* The Relationship between Heart Rate Variability and Adiposity Differs for Central and Overall Adiposity. *Journal of Obesity*, 2012, Article ID 149516.
18. Fentem PH. ABC of Sports Medicine: Benefits of exercise in health and disease. *MJ.* 1994; 308:1291-1295.