ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Study of Sympathovagal Balance in Obese Males.

Chethan HA*, and Priya CR.

Department of Physiology, Subbaiah Medical College, Shimoga, Karnataka, India.

ABSTRACT

Higher incidence of sudden cardiac death indicates possibility of autonomic nervous system imbalance on heart. Increasing number of studies are suggesting that Obesity is one of the major risk factors leading to several cardiac diseases. Studies have shown that autonomic imbalance is one of the leading cause for an increased incidence of sudden cardiac death. This study focuses on using Heart Rate Variability as a tool to determine sympathovagal balance in obese young males [20- 24 years], and also trying to find out if the pattern of distribution of fat in the body has an edge over mere obesity in affecting sympathovagal balance. This study thus might help, as early establishment of this relation and intervention prevents future cardiac autonomic imbalance due to obesity. Subjects were categorized as controls (A) [BMI < 25kg/m^2; n=50], cases (B) [BMI > 30kg/m^2; n=50] based on BMI. Both groups were further divided into three subgroups based on WHR (A1, B1: WHR < 0.83, A2, B2: WHR = 0.84-0.88, A3, B3: WHR = 0.89-0.94). The subjects were informed in advance about the procedures to be done. ECG was recorded in a quiet room with the subject in supine position after 10 minutes of rest. Analog ECG signal was converted to digital by using National Instruments NI-DAQ 7.5 USBD 6008. HRV parameters like SDNN, E:I, LF nu, HF nu, LF/HF were calculated with the help of HRV software (version 1.1). There was a significant ($p<0.01$) decrease in SDNN, E/I ratio, HF nu and a significant ($p<0.01$) increase in LF nu, LF/HF in Obese group (cases) when compared to Normal group (controls). With increasing WHR in both the normal group and obese group, there was no significant change in any of the parameters. The current study showed presence of cardiac autonomic dysfunction in the obese group with the shift of sympathovagal balance towards sympmathetic activity. This study also shows that the amount of fat, rather than its distribution mainly influences the effect of obesity on cardiac autonomic activity among the subjects studied.

Keywords: Body Mass Index, Heart Rate Variability, Waist Hip Ratio, Sympathetic activity.

Corresponding author

ISSN: 0975-8585

INTRODUCTION

With the rising economic standards of people obesity is emerging as important risk factor for health [1]. In addition to high nutritious food, the modern utilities have increased sedentary lifestyle of people hence calorie retention [2]. Of many risk factors, obesity is indeed and important causative factor for multiple conditions leading to metabolic and cardiac disorders [3]. Out of many causes of sudden cardiac death, the correlation between obesity is attaining greater significance [4,5]. Obesity is accompanied with varied combinations of abnormalities in the autonomic nervous system like, obese people have a higher cardiac sympathetic activity that was in some studies been proved to correlate with the catecholamine levels. But there is also evidence of reduced cardiac sympathetic tone in some studies which was partly explained on the duration of obesity [6]. Heart Rate Variability (HRV) is a specific and sensitive noninvasive tool to evaluate cardiac autonomic activity. HRV is the degree of variation of the heart rate under the balanced influence of sympathetic and parasympathetic components of the cardiac autonomic nervous system, and the extent of damage to cardiac autonomic activity [7]. This study is an effort to assess the effect of obesity and also the pattern of distribution of fat in the body on cardiac autonomic activity using Heart Rate Variability in young males as sudden cardiac death in later ages can be prevented if life style modifications can be brought in earlier.

MATERIALS AND METHODS

Healthy male subjects in the age group of 20-24 years comprising 50 normal subjects and 50 obese male subjects were selected from the patient's attendants coming to Sri Siddhartha Medical College Hospital, department of Physiology. After examining, healthy subjects were recruited for the study. Anthropometric parameters like height, weight, waist circumference, hip circumference were recorded. Body Mass Index (BMI) and Waist Hip Ratio (WHR) were calculated [8,9]. In this study both BMI and WHR were considered and the subjects were subdivided into groups as follows [8,9].

They were broadly categorized as

Group A [n=50] - BMI < 25kg/m^2, (**control** group)
Group B [n=50] - BMI > 30kg/m^2, (**cases** group)

Further group A and group B were divided into three subgroups based on WHR

Subgroups of **A** (WHR range): - **A1** (0.83), **A2** (0.84-0.88), **A3** (0.89-0.94)
Subgroups of **B** (WHR range): - **B1** (0.83), **B2** (0.84-0.88), B3 (0.89-0.94)

Subject's clinical history and details were taken according to the standard proforma.

Informed written consent was taken from all subjects in the study.

Inclusion criteria: Healthy males in the age group of 20-24 years were included in the study.

Exclusion criteria: Subjects with major illness like diabetes mellitus, hypertension, respiratory illness, cardiac diseases, recent history of major surgeries and endocrinal disorders were excluded. Subjects on any drugs affecting the functioning of Autonomic Nervous System- adrenergic blockers, calcium channel blockers, anxiolytics, anesthetics, narcotics, chemotherapeutics were also excluded.

Measurement of Heart Rate Variability parameters [7]:

Subject was explained in detail about the ongoing procedure, ECG was digitally recorded after 10 minutes rest using lead II. First, a 5 minutes ECG in supine position was recorded, with subject breathing normally and used to determine the SDNN LF, HF and LF/HF. Then, 2 minutes ECG is taken in a different manner. The person was periodically instructed to take alternating 5 seconds of deep inspiration and 5 seconds of deep expiration for a period of 2 minutes and simultaneous ECG was recorded. This was done to activate the sinus arrhythmia. This data was used to measure the E/I ratio.

Instruments used

- ECG machine (BPL Cardiart 1087/MK-V) was used to acquire ECG signal.
- Analog to digital converter (National Instruments NI-DAQ 7.5 USBD 6008) was used as the hardware, to convert the analog signal to digital and processed it to the computer with the help of the NI-DAQ software.
- Heart Rate Variability software (version 1.1), was used in the computer, to detect the peak to peak intervals and further mathematical and analytical calculations in order to get the values of the parameters.

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean +/- SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. Analysis of variance (ANOVA – version 1.1) has been used to find the significance of study parameters between three or more groups of patients, Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups Inter group analysis). Effect size has been computed to find the effect of obesity on HRV parameters. Student 't' test (two tailed; independent) has been used to test the homogeneity samples based on of age (or continuous parameters).

Parameters [7]: values of SDNN, E/I ratio, HF nu reflects parasympathetic activity
Values of LF nu and LF/HF reflects sympathetic activity.

RESULTS

Basic characteristic features of group A and group B

The controls and cases were age matched (p = 0.155). Whereas, there was a significant increase in BMI (p<0.001), waist circumference (p<0.001), hip circumference (p<0.001) & WHR (p<0.001) in cases when compared to controls [Table No. 1].

Comparison of Heart Rate Variability

SDNN was significantly reduced in cases (p<0.01) [Table No. 2] when compared to controls. There was no significant change of SDNN with increasing WHR, within control group (p=0.823) [Table No. 3] and within cases group (p=0.874) [Table No. 4].

E/I ratio was significantly reduced in cases (p<0.01) [Table No. 2] when compared to controls. There was no significant change of E/I ratio with increasing WHR, within control group (p=0.627) [Table No. 3] and within cases group (p=0.709) [Table No. 4].

LF nu was significantly increased in cases (p<0.01) [Table No. 2] when compared to controls. There was no significant change of LF nu with increasing WHR, within control group (p=0.413) [Table No. 3] and within cases group (p=0.435) [Table No. 4].

HF nu was significantly reduced in cases (p<0.01) [Table No. 2] when compared to controls. There was no significant change of HF nu with increasing WHR, within control group (p=0.413) [Table No. 3] and within cases group (p=0.435) [Table No. 4].

LF/HF was significantly increased in cases (p<0.01) [Table No. 2] when compared to controls. There was no significant change of LF/HF with increasing WHR, within control group (p=0.553) [Table No. 3] and within cases group (p=0.666) [Table No. 4].

ISSN: 0975-8585

Table 1: Comparison of basic characteristics between control and cases

Variables	Controls	Cases	P value
Age in years	21.24±1.47	21.82±1.52	0.155
BMI (kg/m^2)	24.26±0.66	30.64±0.45	<0.001***
Waist circumference (cm)	77.50±4.21	87.92±6.13	<0.001***
HIP circumference (cm)	94.82±4.98	100.98±6.24.	<0.001***
Waist Hip Ratio (WHR)	0.82±0.03	0.87±0.03	<0.001***

Results: mean±SD: *** Very highly significant

Table 2: Comparison of Time Domain and Frequency Domain parameters between controls and cases

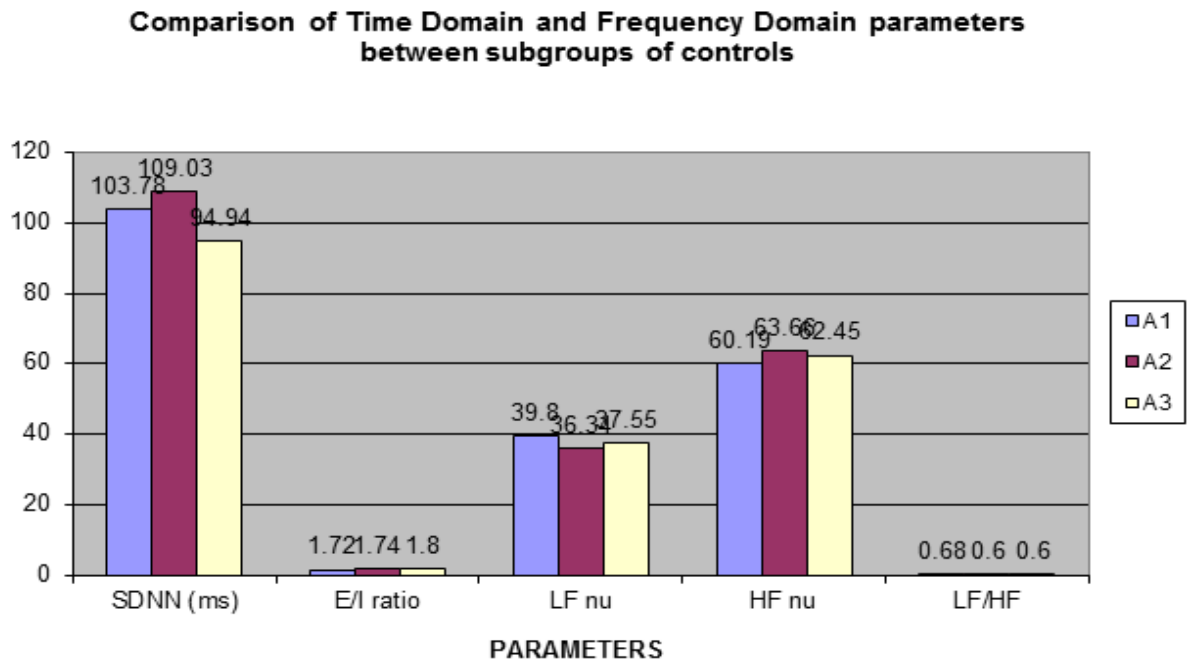
HRV	Controls	Cases	P value	Effect size
SDNN (ms)	104.65±27.86 (69.79-164.37)	40.96±19.18 (12.72-75.19)	<0.01**	2.64
E/I ratio	1.73±0.15 (1.39-1.97)	1.32±0.14 (1.11-1.56)	<0.01**	2.80
LF nu	39.06±7.34 (24.63-54.80)	71.81±9.72 (45.88-87.95)	<0.01**	3.77
HF nu	60.94±7.34 (45.20-75.37)	28.19±9.72 (12.05-54.12)	<0.01**	3.77
LF/HF	0.66±0.27 (0.33-1.21)	3.04±1.63 (0.85-7.30)	<0.01**	2.02

Values Mean ± SD: ** Highly significant

Table 3: Comparison of Time Domain and Frequency Domain parameters between subgroups of controls

HRV	Subgroups of A (WHR range)			p value
	A1 (<0.83)	A2 (0.84-0.88)	A3 (0.89-0.94)	
SDNN (ms)	103.78±29.21	109.03±24.22	94.94	0.823
E/I ratio	1.72±0.16	1.74±0.12	1.80	0.627
LF nu	39.80±6.82	36.34±9.28	37.55	0.413
HF nu	60.19±6.82	63.66±9.27	62.45	0.413
LF/HF	0.68±0.20	0.60±0.24	0.60	0.553

Values Mean ± SD



ISSN: 0975-8585

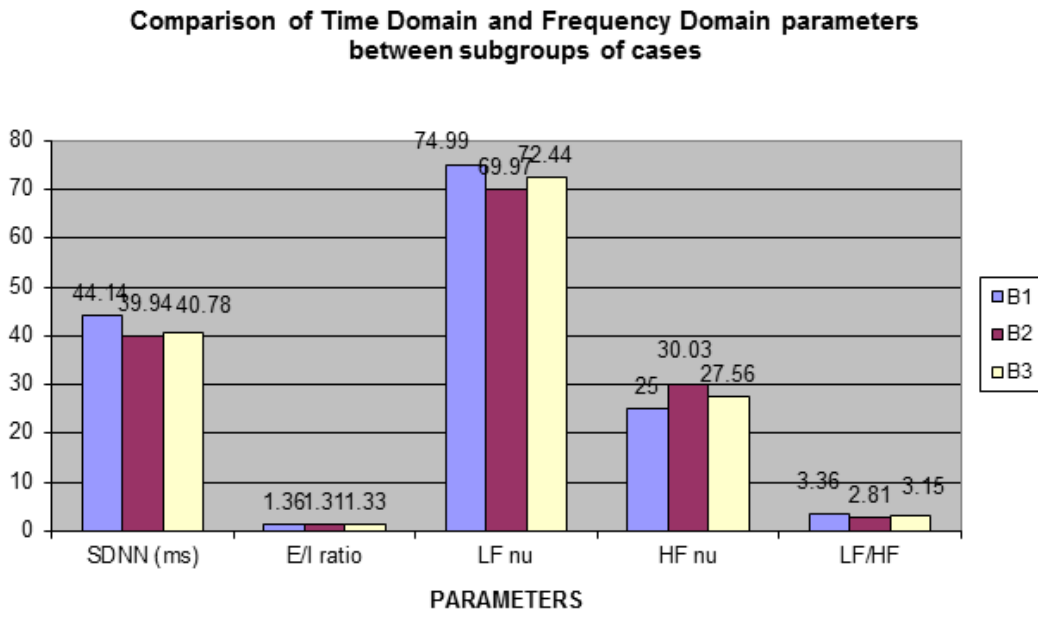


Table 4: Comparison of Time Domain and Frequency Domain parameters between subgroups of cases

HRV	Subgroups of B (WHR range)			p value
	B1 (<0.83)	B2 (0.84-0.88)	B3 (0.89-0.94)	
SDNN (ms)	44.14±17.53	39.94±18.73	40.78±20.93	0.874
E/I ratio	1.36±0.12	1.31±0.14	1.33±0.14	0.709
LF nu	74.99±6.51	69.97±10.42	72.44±10.09	0.435
HF nu	25.00±6.51	30.03±10.42	27.56±10.01	0.435
LF/HF	3.36±1.67	2.81±1.59	3.15±1.69	0.6663

Values Mean ± SD

DISCUSSION

This study aim at assessing the effect of obesity and the pattern of distribution of fat on cardiac autonomic activity in healthy males 20-24 years. The major findings of this study were that the obese group showed significant reduction in the values of SDNN, E/I ratio, HF nu and showed a significant increase in the values of LF nu and LF/HF when compared to normal subjects. Above finding indicate the presence of impaired parasympathetic activity and elevated level of sympathetic activity in obese group. There was a reduction in the parasympathetic activity in obese individuals as shown by reduced SDNN values, E/I values and HF nu values. There was a significant rise in the cardiac sympathetic activity as shown by rise in LF nu values and LF/HF values in the obese group [10-12].

These results of the present study are partly in line with the results of most of the other studies [13-18] and at the same time has different findings compared to other studies [19]. As far as the parasympathetic activity is considered almost all of the studies show a significant reduction in the parasympathetic activity with increasing body weight [13-18]. Contrasting to present study, some studies showed a significant reduction in the sympathetic activity which was partially explained on the basis of the duration of obesity [19]. It has been said that duration of the obesity has a major role to play in determining the level of cardiac sympathetic activity [20].

Sympathovagal balance is the predominance of parasympathetic activity on heart in normal individuals. The shift of sympathovagal balance towards sympathetic activity could very much predispose obese subjects to cardiac rhythm disorders [21].

Present study showed an increase in sympathetic activity in obese individuals, but if the obesity is of a longer duration, then according to some studies it is likely to lead to global reduction of the autonomic activity and hence a reduction in the sympathetic activity also [19].

This study showed significant inverse relationship between BMI and HRV parameters like SDNN, E/I ratio and HF nu, but at the same it showed significant positive relation of BMI and LF nu, LF/HF. The present study shows that the tilt of the sympathovagal balance to sympathetic side and substantial reduction in parasympathetic component is related only to the amount of fat (BMI) but not to the distribution of fat (WHR). This study also showed that there was no significant variation of HRV parameters within the subgroups of control subjects i.e., A1, A2, A3 subdivided based on WHR. There was also no significant variation of HRV parameters within the subgroups of obese subjects i.e., B1, B2, B3 subdivided based on WHR.

A critical question can be raised as to why HRV parameters did not change significantly change with increasing WHR? This lack of consistency can be interpreted as, the further alteration of the HRV parameters did not happen within subgroups of A or subgroups of B where BMI was same. Thus showing the significance of total body fat (BMI) over regional fat distribution (WHR) in causing pathological alterations in cardiac autonomic nervous system functioning. This shows that might be in obese group by the virtue of obesity, there is already a significant alteration in the autonomic nervous system functioning, which provides less scope for WHR to further add on to the existing derangements. But in the normal group as one could expect there should have been an inverse relationship between WHR and HRV parameters, which was not significantly demonstrated in this study, thus suggesting existence of other factors affecting HRV in this group.

Limitations of the present study is that the duration of obesity was not considered in this study, which could have helped in establishing the relation of duration & effects of obesity on cardiac autonomic activity. Serum levels of hormones like catecholamines, leptin etc. could have been considered for better understanding of relation of obesity and its effects on autonomic nervous system.

There is scope for further studies like - study could be undertaken in various age groups and also in both genders for more clarity. Further a prospective study can be undertaken in the same subjects to know the effect of weight loss on the cardiac autonomic activity.

CONCLUSIONS

The present study showed an increase in the cardiac autonomic activity in obese individuals. Obese group also showed a significant reduction of parasympathetic activity, and thus there was a shift in the sympathovagal balance towards sympathetic predominance among obese males, due to increase in the sympathetic activity as a compensatory mechanism of body to reduce excess weight in obese. This sympathovagal imbalance can explain the increased incidence of sudden cardiac deaths associated with obesity. Thus early interventional programs like weight reduction, life style changes and physical exercises, which reduce fat content of the individual, can be advised to reduce the chances of subsequent cardiac rhythm abnormalities [2-6].

In addition, this study also showed that mainly the amount of fat (BMI) rather than its distribution (WHR) influences the effect of obesity on cardiac autonomic activity among the subjects studied, thus stressing on overall weight reduction rather than targeted weight reduction.

ACKNOWLEDGEMENTS

The completion of this research work would never be possible without the guidance, assistance and constant encouragement given to me by a number of people – both teaching staff, technical and non – technical staff of the department of Physiology. Last but not the least the subjects.

REFERENCES

[1] Nageswari SK, Sharma R, Kohli DR. Indian J Physiol Pharmacol 2007; 51(3):235-43.
[2] Younghee K, Youn KS, Haymie C. Obes Res 2004;12: 445-453.
[3] Yuko A, Hironobu Y, Mamoru K, Toshie S, Hiroshi E, Sukenobu I. Exp Biol Med 2001;226(5):440-5.
[4] Kirsten LR, Harry H, Meena K, Eric B, Marek M, Michael M. Am J Epidemiol 2003;158:135-43.
[5] Frenco R, Bernard S, Andrea C, Tiziana G, Barbara DV, Ivana R et al. Obes Rese 2003 April;11(4):541-8.
[6] Jeong AK, Young GP, Kyung HC, Myung HH, Hee CH, Youn SC, Dokyung Y. J Am Board Fam Pract 2005;18:97-103.

[7] European Heart J 1996;17:354-81.
[8] William DM, Katch FI, Katch VL. Essentials of exercise physiology : Body composition, obesity and weight control. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2006. 558-627.
[9] Park K.Text book of preventive and social medicine: Epidemiology of Non-Communicable Disease: Obesity. 19th ed. Jabalpur: Banarsidas publishers Bhanot; 2007. 332-6.
[10] Bernard R. Fundamentals of Biostatistics. 5th ed. Duxbury: 2000. 80-240.
[11] Reddy MV. Statistics for mental health care research. 2nd ed. Delhi: NIMHANS publication; 2002. 108-144.
[12] Troisi RJ, Weiss ST, Parker DR, Sparrow D, Young JB, Landsberg L. Hypertension 1991;17:669 –77.
[13] Emdin M, Gastaldelli A, Muscelli E. Circulation 2001;103:513–9.
[14] Karason K, Molgaard H, Wikstrand J, Sjostrom L. Am J Cardiol 1999;83(8):1242-7.
[15] Chaloupka J, Hlubik P, Opltova L, Mala H. Sb Lek 1998;99(3):251-4.
[16] Martini G, Riva P, Rabbia F, Molini V, Ferrero GB, Cerutti F et al. Clin Auton Res 2001;11(2):87-91.
[17] Facchini M, Malfatto G, Sala L, Silvestri G, Fontana P, Lafortuna C et al. J Endocrinol Invest 2003 Feb;26(2):138-42.
[18] Poirier P, Hernandez TL, Weil KM, Shepard TJ, Eckel RH. Obes Res 2003 Sep;11(9):1040-7.
[19] Masuo K, Mikami H, Ogihara T, Tuck ML. Hypertension 2000;35:1135-40.
[20] Nagai N, Moritani T. Int J Obes Relat Metab Disord 2004 Jan;28(1):27-33.
[21] Hnatkova K, Copie X, Staunton A, Malik M. J Electrocardiogr 1995;28:74–80.