Research Article

COMPARISON OF AUTONOMIC CARDIOVASCULAR FUNCTIONS USING HEART RATE VARIABILITY TEST IN YOUNG OBESE AND NON OBESE MALES AND FEMALES

*Khwaja Nawazuddin Sarwari¹, Moin Sabeer², Mohammed Imran³ and Arshiya Zeba⁴

¹Department of Physiology, Khaja Banda Nawaz Institute of Medical Sciences, Gulbarga, Karnataka

²Department of Biochemistry, Khaja Banda Nawaz Institute of Medical Sciences, Gulbarga, Karnataka

³Department of Community Medicine, Dr.B.R.Ambedkar Medical College, Bangalore, Karnataka

⁴Department of Physiology, Bidar Institute of Medical Sciences, Bidar, Karnataka

*Author for Correspondence

ABSTRACT

Obesity is often defined simply as a condition of abnormal or excessive fat accumulation in adipose tissue, obesity produces an increment in total blood volume and cardiac output that is caused in part by the increased metabolic demand induced by the excess body weight, since cardiovascular system have been reported to be the one of the major system reflecting the adverse effects of obesity, the present study is undertaken to assess the cardiovascular autonomic functions using Heart rate variability (HRV) in obese young Males and Females and compare the results with the controls.

Key Words: Obesity, HRV, Cardiovascular Autonomic Functions

INTRODUCTION

Jousilahti *et al.*, (1996) reveals that Overweight and obesity may account for as many as 15 - 30% of deaths from coronary heart disease (CHD) and 65-75% of new cases of type 2 diabetes mellitus.

Hotamisligil *et al.*, (1995) suggests that the adipose tissue is not simply a passive storehouse for fat but an endocrine organ that is capable of synthesizing and releasing into the blood stream an important variety of peptides and non-peptide compounds that may play a role in cardiovascular homeostasis. Adipose tissue is a significant source of Tumor Necrosis Factor-alpha (TNF- α), interleukin-6, plasminogen activator inhibitor-1, leptin, angiotensinogen and insulin like growth factor – 1 (IGF-1).

Alpert *et al.*, (2001) observed that Obesity produces an increment in total blood volume and cardiac output that is caused in part by the increased metabolic demand induced by excess body weight. The increase in blood volume in turn increases venous return to the heart, increasing filling pressures in the ventricles and increasing wall tension. This leads to left ventricular hypertrophy and this can decrease the diastolic compliance of the ventricle which can further, can lead to systolic dysfunction.

T Sai et al., (2002) and Blaszyk et al., (1999) reveals that the incidence of venous thromboembolism in the upper tertile of BMI was 2.42 times that in the lowest BMI tertile, and waist circumference > 100 cm in men was also related to venous thromboembolism, morbid obesity is an independent risk factor for death from pulmonary embolism.

Stamler *et al.*, (1978) suggests that the hypertension is about 6 times more frequent in obese subjects than in lean men and women. Weight gain in young people is a potent risk factor for subsequent development of hypertension. The increase in blood pressure is greatest when the obesity is of abdominal distribution. Benjamin *et al.*, (1999) and Hirsch *et al.*, (1991) reveals that the activation of the sympathetic nervous system occurs early in the course of obesity and the autonomic nervous system is an important contributor to the regulation of both the cardiovascular system and energy expenditure. A 10% increase in body weight is associated with a decline in parasympathetic tone accompanied by a rise in mean heart rate and conversely, heart rate decline during weight reduction. Fluctuation of heart the rate around mean heart rate provides information on the activity of the cardiac autonomic system which is called Heart Rate Variability (HRV).

Rebkin et al., (1983) observed that the obese subjects have an increased risk of arrthythmias and

Research Article

sudden death even in the absence of cardiac dysfunction and the risk is about 40 times higher than in matched non-obese population in the Framingham Heart study.

MATERIALS AND METHODS

60 obese and 60 non-obese young males and females of the age group 18-25 years were selected randomly from the general population of Gulbarga city (students, healthy attendants of patients of KBN Hospital).

Inclusion criteria:

- Young obese males and female aged 18-25 years.
- Young non obese males and females aged 18-25 years.

Exclusion criteria:

- Age below 18 years and above 25 years.
- Subjects with history of Asthma, Diabetes, Mellitus, Hypertension, other cardiovascular diseases, endocrine disease or surgery.
- Subjects on chronic medication.
- Smokers
- Alcoholics
- Subjects with noticeable weight loss over the preceding 3 month
- Subjects having any neuro-muscular disorders.

The benchmark for obesity was taken on the basis of body mass index as per the standard protocol. Height (m) and weight (kg) of the subjects will be recorded and BMI calculated as per Quetelet's index

Body mass index =	Weight (kilogram
Body mass macx –	Height ² (meter)

Subjects are classified into 2 groups based on BMI as follows:

Normal weight $- BMI - 18.5 - 24.99 \text{ kg/m}^2$

And / obese $-BMI > 30 \text{ kg/ } \text{m}^2$

A pretested structured proforma is used to collect the relevant information. All subjects were explained about the procedures to be undertaken and written informed consent was obtained from them.

Power spectral analysis of heart rate variability test is performed 2-3 hours after light breakfast after familiarizing the subjects with the testing procedure.

Frequency domain analysis was done by using Niviqure software. Analysis of heart rate variability was assessed by spectral analysis of series of successive R.R. interval (frequency domain analysis) on 5 min EGG recording. The heart rate power spectrum is typically divided into two frequency bands. Low $(0.04 - 0.15 \, \text{Hz})$ and high $(0.15 \, \text{to} \, 0.4 \, \text{Hz})$. The high frequency region is generally considered a marker of vagal activity where as the low frequency component influenced by both sympathetic and vagal activity.

RESULTS AND DISCUSSION

60 obese (30 males and 30 females) subjects and 60 non obese (30 males and 30 females) subjects were analysed for the results. The results obtained are expressed as mean + standard deviation.

On analysis of the physical characteristic of the 30 non obese females, the mean age (years) is 19.8 ± 2 ; the mean weight (kg) is 54.27 ± 6.33 ; the mean height (mt) is 1.59 ± 0.05 ; the mean BMI (kg/m²) is 21.43 + 1.63 (Table 1).

On analysis of the physical characteristics of the 30 obese male subjects, the mean age (years) is 21.2 ± 1.8 ; the mean weight (kg) is 80.90 ± 7.23 ; the mean height (mt) is 1.59 ± 0.08 ; the mean BMI (kg/m²) is 32.13 ± 1.71 (Table 1).

Research Article

On analysis of the physical characteristics of the 30 obese female subjects, the mean age (years) is 20.5 ± 2.0 ; the mean weight (kg) is 83.4 ± 5.82 ; the mean height (mt) is 1.61 ± 0.06 ; the mean BMI (kg/m²) is 32.33 ± 1.81 (Table 1).

Heart rate variability:

Total power (TP):

The mean value of total power (ms²) in non-obese males was 1881.3 ± 443.6 and in obese males was 1284.5 ± 154.4 . There was a statistically significant decrease in total power (p < 0.001) in obese males as compared to non-obese males (Table 2).

The mean value of total power (ms²) is non-obese females was 1692.7 ± 382 and is obese females was 1373.1 ± 338.1 . There was a statistically significant decrease in total power (p < 0.001) in obese females as compared to non obese females (Table 3).

LF Power (ms²):

The mean value of LF power (ms²) in non obese males was 826.5 ± 74.2 and in obese males was 785.4 ± 69.6 . There was a statistically significant (p < 0.005). Decrease in LF power in obese males as compared to non obese males (Table 2).

The mean value of LF power (ms²) in non obese females was 808.5 ± 74.2 and in obese females was 758.4 ± 69.6 . There was a statistically significant decrease in LF power in obese females (p < 0.001) as compared to non-obese females (Table 3).

HF power (ms^2) :

The mean value of HF power (ms²) in non-obese males was 1054.8 ± 417.7 and in obese males was 499.1 ± 132 . There was a statistically significant decrease in HF power in obese males (p < 0.001) as compared to non obese males (Table 2).

The mean value of HF power (ms²) in non-obese females was 884 ± 354.4 and in obese females was 614.7 ± 304.3 . There was a statistically significant decrease in HF power in obese females (p < 0.001) as compared to non obese females (Table 3).

LF power in normalized units (n.u):

The mean value of LF (n.u) in non-obese males was 45.8 ± 6.9 and in obese males was 64.6 ± 9.0 . There was a statistically significant increase in LF (n.u) in obese males (p < 0.001) as compared to non-obese males (Table 2).

The mean value of LF (n.u) in non-obese females was 45.1 ± 8.7 and in obese females was 53 ± 8.3 . There was a statistically significant increase in LF (n.u). in obese female (p<0.001)as compared to non-obese females (Table 3).

HF power in normalized units (n.u):

The mean value of HF (n.u.) in non obese males was 33.6 ± 4.1 and in obese males was 20 ± 3.6 . There was a statistically significant decrease in HF (n.u) in obese males (p<0.001) as compared to non obese males (Table 2).

The mean value of HF (n.u) in non-obese females was 33.5 ± 6.0 and in obese females was 175 ± 4.6 . There was a statistically significant decrease in H.F (n.u) in obese females (p<0.001) as compared to non-obese females (Table 3).

LF/HF ratio:

The mean value of LF/HF in non obese males was 0.91 ± 0.37 and is obese males was 1.65 ± 0.35 . There was a statistically significant increase in LF/HF in obese male (p<0.001) as compared to non obese males (Table 2).

The mean value of LF/HF in non obese females was 1.05 ± 0.38 and in obese females was 1.45 ± 0.52 . There was a statistically significant increase in LF/HF in obese females (p<0.01) as compared to non obese females (Table 3).

Research Article

Table 1: Physical characteristics of the subjects

		Males		Females		Total	_
No. of Subjects		Controls	Obese	Controls	Obese	Controls	Obese
		30	30	30	30	60	60
	Mean <u>+</u>	21.0 <u>+</u>	21.2 <u>+</u>	19.8 <u>+</u>	20.5 <u>+</u>	20.4 <u>+</u>	20.9 <u>+</u>
Age	SD	2.3	1.8	2.0	2.0	2.2	1.9
(yrs)	Range	18-25	18-25	18-25	18-25	18-25	18-25
Weight	Mean <u>+</u>	60.80 <u>+</u>	80.90 <u>+</u>	54.27 <u>+</u>	83.40 <u>+</u>	57.5 <u>+</u>	82.2 <u>+</u>
(kg)	SD	5.05	7.23	6.33	5.82	6.6	6.6
Height	Mean <u>+</u>	1.67 <u>+</u>	1.59 <u>+</u>	1.59 <u>+</u>	1.61 <u>+</u>	1.63 <u>+</u>	1.60 <u>+</u>
(mt)	SD	0.05	0.08	0.05	0.06	0.07	0.07
	Mean <u>+</u>	21.70 <u>+</u>	32.13 <u>+</u>	21.43 <u>+</u>	32.33 <u>+</u>	21.67 <u>+</u>	32.23 <u>+</u>
BMI	SD	1.73	1.71	1.63	1.81	1.67	1.75
	Range	18.4-	30.1-	18.5-	30.1-	18.4-	30.1-
		24.5	36.6	24.9	36.4	25.0	36.6

Table 2: Comparison of HRV between obese and non obese Males

	Control males		Obese mal	Obese males		Control vs obese	
	Mean	SD	Mean	SD	T value	P level	
TP (ms2)	1881.3	443.6	1284.5	154.4	6.96	<0.001,HS	
LF (ms ²)	826.5	74.2	785.4	69.6	2.21	0.03	
HF (ms2)	1054.8	417.7	499.1	132.0	6.95	<0.001,HS	
LF (n.u)	45.8	6.9	64.6	9.0	9.12	<0.001,HS	
HF (n.u)	33.6	4.1	20.0	3.6	13.61	<0.001,HS	
LF/HF	0.91	0.37	1.65	0.35	8.02	<0.001,HS	

^{*}unpaired 't' test

Table 3: Comparison of HRV between obese and non obese Females

	Control females		Obese fem	Obese females		Control vs obese	
	Mean	SD	Mean	SD	T value	P level	
TP (ms2)	1692.7	382.0	1373.1	338.1	3.43	0.001	
LF (ms ²)	808.5	74.2	758.4	69.6	2.7	0.009	
HF (ms2)	884.2	354.4	614.7	304.3	3.16	0.003	
LF (n.u)	45.1	8.7	53.0	8.3	3.61	0.001	
HF (n.u)	33.5	6.0	17.5	4.6	11.49	<0.001,HS	
LF/HF	1.05	0.38	1.45	0.52	3.40	0.001	

^{*}unpaired 't' test

The main goal of the study is to detect changes of autonomic cardiovascular regulation in healthy obese young adult males and females in the age group 18-25 years and to compare the results with the age and sex matched healthy non-obese controls.

The differences in the mean values of each parameter between obese and non-obese males and females are analysed and discussed.

NS – Not Significant

S-Significant

HS – *Highly significant*

NS – Not Significant

S – Significant

HS – *Highly significant*

Research Article

It is evident from the study that healthy young obese males and females have increased resting sympathetic activity as evidenced by increase in L.F. (n.u) and baseline blood pressure as compared with healthy young non obese males and females controls respectively.

There was a decrease in the parasympathetic activity in healthy young obese males and females as evidenced by decrease in HF, HF (n.u) and baseline blood pressure where compared with healthy young non-obese males and females controls respectively.

LF/HF ratio which indicates sympatho-vagal balance is increased in young obese males and females compared with non-obese males and females controls respectively indicating obesity is associated with cardiovascular autonomic imbalance.

The following conclusions can be drawn from the results of the present study.

- An increase in the body weight is associated with an increase in systolic and diastolic blood pressure
 in both males and females at resting condition. When compared with age and sex matched normal
 weight controls.
- Low frequency power when expressed in normalized units is increased in obese males and females
 when compared with controls indicating that there is an increase in the cardiac sympathetic activity at
 resting condition in obese subjects.
- High frequency power expressed in both absolute units (ms²) and in normalized units is decrease in obese males and females when compared with controls indicating there is a decrease in the cardiac parasympathetic activity at resting condition in obese subjects.
- Low frequency to high frequency power ratio increased in obese males and females when compared with controls indicating there is a sympatho-vagal imbalance in obese subjects.
- Though there is an increase in resting sympathetic activity in obese males and females as evidenced by increase in baseline blood pressure and LF (n.u) component of HRV, but the sympathetic response after isometric exercise is reduced in obese males and females.

ACKNOWLEDGEMENT

The author thanks the Head, Department of Physiology for his suggestions. Special thanks to Principal, Khaja Banda Nawaz Institute of Medical Sciences for his encouragement.

REFERENCES

Alpert MA (2001). Obesity cardiomyopathy; pathophysiology and evolution of the clinical syndrome. *The American Journal of Medical Science* **321**(4) 225-236.

Benjamin EJ, Levy D (1999). Why is left ventricular hypertrophy so predictive of morbidity and mortality? *The American Journal of Medical Science* 317(3) 168-175.

Blaszyk H, Wollan PC, Witkiewicz AK, Bjornsoon J (1999). Death from pulmonary thromboembolism in severe obesity: lack of association with established genetic and clinical risk factors. *Virchows Archiv* **434**(6) 529-532.

Hirsch J, Leibel RL, Mackintosh R, Aguirre A (1991). Heart rate variability as a measure of autonomic function during weight change in humans. *American Journal of Physiology* **261** 1418-23.

Hotamisligil GS, Arner P, Caro JF, Atkinson RL, Spiegelman BM (1995). Increased adipose tissue expression of tumor necrosis factor-alpha in human obesity and insulin resistance. *The Journal of Clinical Investigation* 95(5) 2409-2415.

Jousilahti P, Tuomilehto J, Vartiainen E, Pekkanen P (1996). Body weight, cardiovascular risk factors, and coronary mortality: 15 – year following-up of middle-aged men and women in Eastern Finland. *Circulation* 93 1372-79.

Rabkin SW, Mathewson FA, Hsu PH (1977). Relation of body weight to development of ischemic heart disease in a cohort of young North American men after a 26 year observation period: the Manitoba Study. *American Journal of Cardiology* **39**(13) 452-458.

Research Article

Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH (1978). Weight and blood pressure: findings in hypertension screening of 1 million Americans. *The Journal of the American Medical Association* **240**(15) 1607-10.

Tsai AW, Cushman M, Rosamond WD, Heckbert SR, Polak JF, Folsom AR (2002). Cardiovascular risk factors and venous thromboembolism incidence: the longitudinal investigation of thromboembolism etiology. *Archives of Internal Medicine* **162**(10):1182-89.