

Heart Rate Variability Analysis in Young Obese Individuals

M.Sivaraj¹, M.Janet Sugantha¹

¹Associate Professor, Department of Physiology, K.A.P.V.Govt. Medical College, Tiruchirapalli

Abstract

Introduction: Obesity is associated with marked cardiorespiratory changes. In obese individuals there occurs an increase in heart rate, systolic blood pressure and cardiac output up to 15%. These changes are thought to occur as a result of changes in autonomic activity. Among the available noninvasive techniques for assessing autonomic status, heart rate variability has emerged as a simple non-invasive method to evaluate sympatho-vagal balance.

Materials and Method: Heart Rate Variability analysis was done in 30 young obese individuals and compared with 30 age and gender matched individuals with normal BMI

Results: In obese individuals, the SDNN was significantly higher than in controls. LF and LF/HF values of obese individuals were significantly higher than those of controls but their HF values were lower.

Conclusion: HRV analysis shows sympathetic overactivity and parasympathetic blunting in young obese individuals.

Keywords: Obesity, Autonomic neuropathy, Heart rate variability

Introduction

Obesity is associated with marked cardiorespiratory changes. In obese individuals there occurs an increase in heart rate, systolic blood pressure and cardiac output up to 15%. These changes which are most likely marked in extreme degree of obesity are thought to occur as a result of changes in autonomic activity. Data on autonomic function in obese people have revealed that they have an increased sympathetic discharge. It is possible that this higher level of sympathetic activity and cardiovascular load is associated with increased platelet aggregation, plaque rupture, coronary vasospasm and explains why the incidence of myocardial infarction and ischaemic strokes is higher in the obese individuals. Patients with increased body mass index are exposed to recurrent episodes of tachycardia, hypoxemia and acute haemodynamic stress and it would seem plausible

that these factors are instrumental in the development of essential hypertension or other cardiovascular diseases from clinical study. There is nevertheless mounting experimental evidence that patients with obesity have higher level of autonomic activity especially of the sympathetic pattern. Juan Sztajzel et.al., (2004) stated that among the different available noninvasive techniques for assessing autonomic status, heart rate variability has emerged as a simple non-invasive method to evaluate sympatho-vagal balance at the sino atrial node.

Aim of the Study: To evaluate Cardiovascular Autonomic Functions in obese individuals using Heart rate variability analysis

Materials and Method

CASES: 30 obese individuals in the age group of 18 to 25 years, without co-morbid conditions like Diabetes, Hypertension, Smoking, Alcoholism, Thyroid disorders

WHO criteria was applied to categorize the subjects; Controls – subjects with BMI 18.5 – 24.9 and Obese when their BMI is more than 25

Corresponding author:

Dr. M.Janet Sugantha,

Associate Professor, Department of Physiology,
K.A.P.V.Govt. Medical College, Tiruchirapalli
Email id: janet.sugantha@gmail.com

Controls

30 age and gender matched healthy, non-obese subjects attending the Master health check up programme, Madras Medical College & Government General hospital, Chennai.

The study protocol was approved by the Ethical committee of Madras Medical College. Informed and written consent was obtained from the subjects.

The subjects were instructed to lie down in supine posture and relax for 5 minutes. Resting Heart rate and blood pressure were recorded. By applying 3 electrodes, in the standard positions, HRV was recorded in the supine posture using ECG recorder. The leads were connected to the ECG recorder which in turn was connected by signal processing unit to the computer. The recording was made for 5 minutes. After screening the data for artifacts and properly editing it the data was opened

through HRV analysis software version 1.1 to obtain converted ECG signal. The analog to digital conversion of the resting ECG signal was done using AD converter with sampling frequency of 1024/sec. Power spectral analysis of the converted ECG signal was done using Fast Fourier transformation.

Mean RR, SDNN, Mean HR, Power, LF, HF and LF/HF were estimated.

Results

Statistical Package for Social Sciences (SPSS) software 11.5 version was used for statistical analysis.

The Student independent unpaired 't' test was used .

$p < 0.05$ is taken as significant ;

$p < 0.01$ is taken as highly significant

TABLE 1: Anthropometric measurements and HRV parameters

VARIABLES	CONTROLS	OBESE	p value
AGE in years	21.76 ± 4.25	19.37±3.99	0.13
BMI Kg/m ²	22.25 ± 1.09	35.23±5.36	< 0.001
HR	74.57 ± 5.78	116.87±4.21	< 0.001
MEAN RR	853.33± 70.54	841.53±50.17	0.45
SDNN	54.1 ± 19.65	85.4±8.05	< 0.001
LF	44.66 ± 8.13	60.71±9.93	< 0.001
HF	55.21 ± 8.22	19.47±2.37	< 0.001
LF/HF	0.86 ± 0.31	3.17±0.67	< 0.001

Discussion

The age group of controls and subjects were similar emphasising there were no age related differences in obese individuals and normal people.

The young obese individuals had a significantly higher body mass index, implying they were relatively obese compared to controls.

Robert Wolk et al ⁽¹⁾, suggested that there is a link between obesity and hypertension as well as cardiovascular associated mortality.. They postulated that obese individuals are predisposed to an increased risk of developing hypertension, and treatment of obesity lowers blood pressure and cardiac related morbidity . Possible mechanisms whereby obesity may contribute to hypertension in individuals include sympathetic activation, hyperleptinemia, insulin resistance, elevated angiotensin II and aldosterone levels, oxidative and

inflammatory stress, endothelial dysfunction, impaired baroreflex function, and perhaps by effects on renal function. The existence of high grades of obesity may have more widespread implications for cardiovascular control and dysfunction in these obese individuals and may contribute to some of the clustering of abnormalities broadly defined as the metabolic syndrome. From the clinical and therapeutic perspectives, the presence of resistant hypertension and the absence of a nocturnal decrease in blood pressure in obese individuals should prompt the clinician to consider the diagnosis of the individual suffering from high graded complication of obesity.

Heart Rate Variability (HRV)

HRV is a very valuable tool by itself for risk stratification in cardiovascular diseases. It assesses the autonomic tone at rest. HF component is generally defined as a marker of vagal modulation. This component is respiratory mediated and thus is determined by the frequency of breathing. The LF component is modulated by both sympathetic and parasympathetic activity. LF/HF ratio reflects global sympatho-vagal balance ⁽²⁾

Heart rate variability (HRV) is mediated by at least three primary mechanisms:

Vagal feedback from Pulmonary Stretch Receptors (PSR),

Central medullary coupling between respiratory and cardiovascular neurons (RCC)

Arterial baroreflex (ABR)-induced fluctuations.

In our study there was significant increase in LF ($P < 0.001$), whereas there was an equally significant drop in HF ($P < 0.001$) suggesting a sympathetic overactivity and parasympathetic blunting. The LF/HF ratio also showed a significant increase ($P < 0.001$) indicating a strong sympathetic activity.

Robin Smith et al.⁽³⁾, has shown that patients with obesity have high levels of sympathetic activity during normal activities than the normal weight individuals in whom sympathetic activity generally found to be normal. Pathologically high levels of sympathetic activity persist in these obese individuals. Because of these high sympathetic activity these people have more anxiety sweating, change in mood, depressive episodes etc when compared to normal individuals. (Mcmaillan et al

..2001).. This sympathetic overactivity leads to increase in blood pressure, injury to the major blood vessels of the heart and other organs which may lead to thrombosis and hypercoagulation. Damage to the nervous system may lead to disturbed autonomic functions there by poor regulation of blood pressure and heart rate.

Improper heart rate regulation

Persistent high blood pressure

Blunted cardiac autonomic responses.

Mustafa Aydin et al.⁽⁴⁾, in his study identified that in both mild and severe obesity, SDNN was significantly lower than in controls. LF and LF/HF values of obese individuals were significantly higher than those of controls. Their HF values were lower which was similar to our study.

Power spectrum analysis of heart rate variability in obese patients by Ferini-Strambi L et al.⁽⁵⁾ and Noda et al.⁽⁶⁾, have shown a significant decrease in mean HF and an equally significant increase in LF/HF ratio implying a parasympathetic blunting and an increased sympathetic activity as seen in our study.

HRV is altered in obesity. Respiration, heart rate, and blood pressure were monitored in eight normal subjects and nine increased BMI young adults. The findings of this study suggest that the adverse autonomic effects of obesity include impairment of baroreflex gain and central respiratory-cardiovascular coupling, but the component of respiratory sinus arrhythmia that is mediated by lung vagal feedback remains intact.

Cardiovascular variability is altered in patients with obesity. This alteration is evident even in the absence of hypertension, heart failure, or other disease states and may be linked to the severity of obesity. Abnormalities in cardiovascular variability may be implicated in the subsequent development of overt cardiovascular disease in patients with obesity.

Conclusion

From our study there is clear evidence of sympathetic overactivity and parasympathetic blunting in young obese individuals. Heart rate variability is a simple, non-invasive test to identify Cardiac autonomic dysfunction and it can be used as a screening test in these individuals.

Conflict of Interest: Nil

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